A Dissertation on

A STUDY ON END ORGAN DAMAGE IN NEWLY DETECTED HYPERTENSIVE PATIENTS

Dissertation Submitted to

THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

With partial fulfillment of the regulations

For the award of the degree of

M.D. GENERAL MEDICINE

BRANCH-I

COIMBATORE MEDICAL COLLEGE

COIMBATORE

APRIL 2019

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

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Dissertation Topic	:	A study on end organ damage in newly detected hypertensive patients.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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DECLARATION

I solemnly declare that the dissertation titled "**A STUDY ON END ORGAN DAMAGE IN NEWLY DETECTED HYPERTENSIVE PATIENTS"** Was done by me from JUNE 2017 to JUNE 2018 under the guidance and supervision of Professor **DR.M.RAVEENDRAN M.D** This dissertation is submitted to **The Tamilnadu Dr.M.G.R. Medical University** towards the partial fulfilment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place: Coimbatore **DR.M. MANGAI SUSEELA**

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ACKNOWLEDGEMENT

I wish to express my sincere thanks to our respected **Dean Dr. B. ASHOKAN M.S., MCH** for having allowed me to conduct this study in our hospital.

I express my heartfelt thanks and deep gratitude to the Head of the Department of Medicine Professor. **Dr. KUMAR NATARAJAN, M.D**. for his generous help and guidance in the course of the study.

I express my heartfelt thanks and deep gratitude to my guide **PROF.DR.M.RAVEENDRAN.M.D** for her support and guidance for the study.

I sincerely thank all professors and Asst. Professors- **Dr. P.S.MANSHUR,**

Dr. P. SANBAKASREE, Dr. K. SANGEETHA for their guidance and kind help.

My sincere thanks to Department of BIOCHEMISTRY AND OPHTHALMOLOGY for their help.

My sincere thanks to all my friends and post-graduate colleagues for their whole hearted support and companionship during my studies.

I thank all my **PATIENTS**, who formed the backbone of this study without whom this study would not have been possible.

Lastly, I am ever grateful to the **ALMIGHTY GOD** for always showering His blessings on me and my family.

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This is to certify that this dissertation work titled "**ASTUDY ON END ORGAN DAMAGE IN NEWLY DETECTED HYPERTENSIVE PATIENTS ”** of the candidate DR.M.MANGAI SUSEELA with registration Number 201611305 for the award of M.D in the branch of General Medicine I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 4% of plagiarism in the dissertation.

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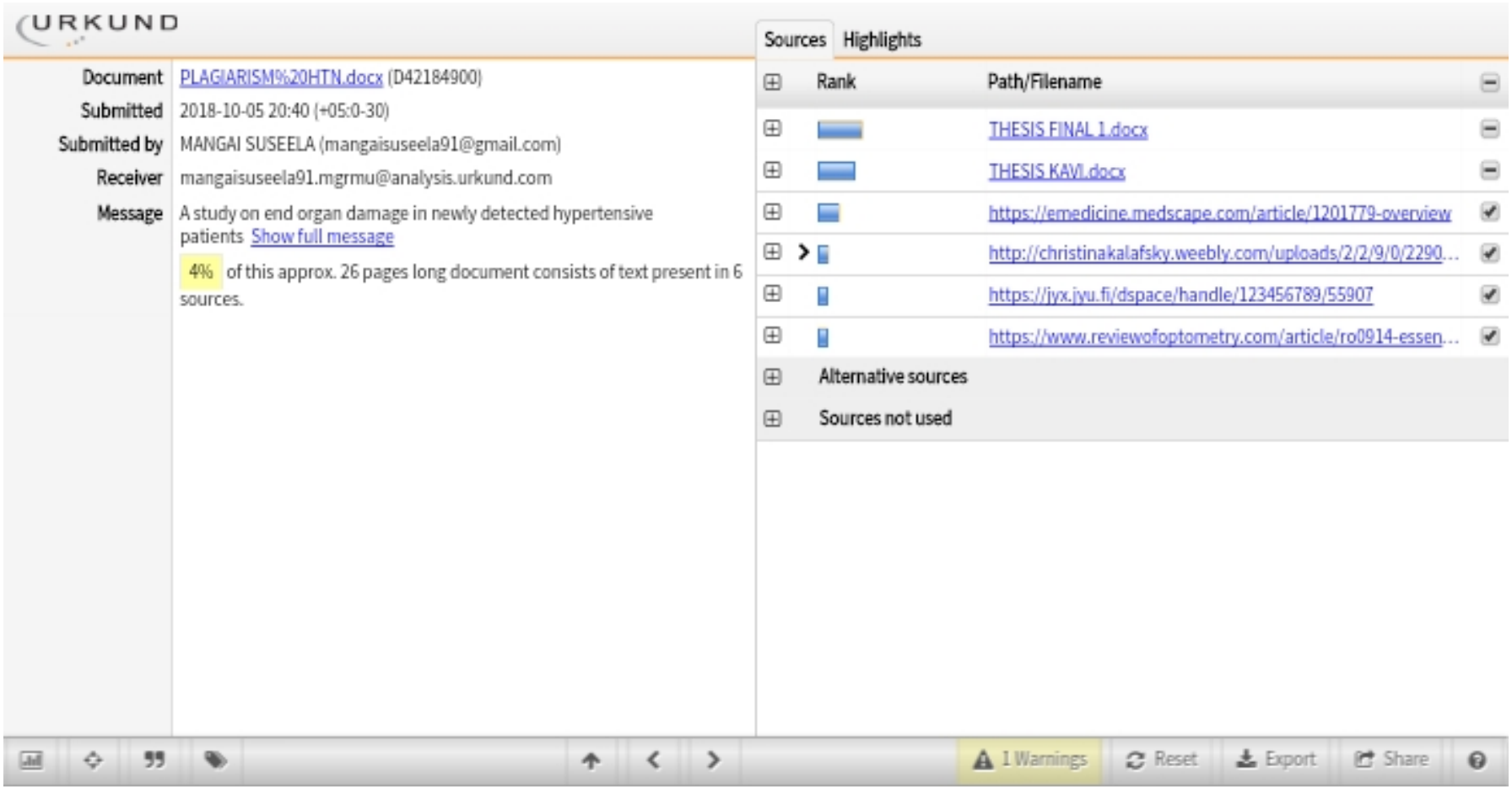
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AIMS AND OBJECTIVES OF STUDY

1. To assess the prevalence of target end organ damage in newly detected hypertensive patients

2. To analyse the severity of hypertension at the time of diagnosis based on target organ damage

INTRODUCTION

Dissertation is an in depth study of a particular topic. My topic is hypertension. Hypertension is a silent killer disease. It is an independent risk factor for coronary artery disease, stroke and renal failure. If diagnosed early, occurrence of complications can be prevented. Prevalence of hypertension is 159.66 per 1000 population in India according to the NCD programme. Globally 17.3 million people died from cardiovascular disease in 2008 representing 30% of all global deaths. Prevalence of hypertension is 21% in rural Tamil Nadu and 22 to 30% in urban Tamil Nadu.

Hypertension is a major health problem and an important risk factor for stroke, coronary artery disease and renal failure. Target organ damage assessment in hypertension is a better predictor of cardiovascular risk in hypertensive patients. It has also significant prognostic significance. The prevalence of hypertension is high in the general population. Adequate treatment of hypertension can reverse and prevent the progression of target end organ damage. Newly detected hypertensive patients can have evidence of target organ damage at the time of diagnosis of the disease. Based on that progression of complications of the disease can be predicted. It also helps in early treatment of target end organ damage.

This study focuses on the target end organ damage in 200 newly detected hypertensive patients attending NCD out-patient department at Coimbatore Medical College Hospital for a period of one year

REVIEW OF LITERATURE

Blood pressure (BP) is the force that the blood exerts on the vessel wall and is continuously varying in arteries due to the intermittent nature of the pump (heart) and elastic recoil of the arterial wall (1). Besides the simple extremes of pressure, i.e. systolic and diastolic, there are 2 major physiological components of the arterial BP:

1. Static or 'steady-state'- Represented by the mean arterial pressure. The main determinants of the mean arterial pressure are cardiac output and peripheral vascular resistance (PVR)

Mean pressure = cardiac output x PVR

2. Pulsatile: represented by the pulse pressure (difference between systolic and diastolic blood pressure). The principal determinants of pulse pressure are the stroke volume and the stiffness of the large arteries.

It affects individuals of all age groups. Hypertension is a leading risk factor for morbidity and mortality throughout the world. The manifestations of hypertensive end organ damage include stroke, retinopathy, coronary heart disease and heart failure, proteinuria and renal failure, atherosclerotic changes including the development of stenosis and aneurysms. The chance of myocardial infarction, heart failure, stroke, and kidney disease is increased as BP is increased. Hypertension remains undiagnosed for a long time so that a large number of hypertensive patients have end organ damage at the time of diagnosis of the disease itself. Accurate diagnosis hypertension remains a

challenge because of other conditions like white coat hypertension, masked hypertension. 24 hour ambulatory blood pressure is considered the best method for diagnosis of hypertension because it eliminates white coat and masked hypertension. It also measures the cardiovascular load.

American Heart Association Guidelines for diagnosis of hypertension: (2)

BLOOD PRESSURE CATEGORY	**SYSTOLIC BLOOD PRESSURE**		**DIASTOLIC BLOOD PRESSURE**
NORMAL	LESS THAN 120	AND	LESS THAN 80
ELEVATED	120-129	AND	LESS THAN 80
STAGE 1 HYPERTENSION	130-139	OR	80-89
STAGE 2 HYPERTENSION	140 OR HIGHER	OR	90 OR HIGHER

Other definitions:

White coat hypertension: Elevated clinic BP and normal home BP; White-coat hypertension is estimated to affect at least 10% of the population, with some estimates suggesting a prevalence of greater than 20% of the population. The condition generally occurs in patients with mild hypertension and is most often seen in the young or in the elderly and is least common in

middle-aged adults. White-coat hypertension contributes to resistant (or refractory) hypertension, which occurs in a small subset of hypertensive patients who do not show a satisfactory reduction of BP even under treatment with a combination of three different hypertension medications.

Masked hypertension is defined as normal clinic BP and elevated home BP. It is important because masked hypertension is associated with increased mortality and morbidity when compared to general population.

Equipment used for blood pressure measurements:

Auscultation of the Korotkoff sounds using a sphygmomanometer remains the most common method for checking BPs during clinic visits. Mercury sphygmomanometers are most accurate, but due to cost and the potential chemical hazards of mercury, aneroid gauges are more often used. For accuracy of measurement aneroid devices should be calibrated every six months against a mercurycontaining sphygmomanometer (3). Therefore, alternative devices, which rely on an automated oscillometer, are being increasingly used for BP measurement in clinics and at home. Automated methods have the advantage of reduced operator error and they require less training. However, automated oscillometers have greater inherent errors and are considered inaccurate for careful BP measurement. Oscillometers also generally record lower BP values for a patient compared with a sphygmomanometer, making comparison between the methods more difficult. Using a correct cuff size is crucial for accurate BP measurements. The bladder

contained in the cuff should be long enough to cover 80% of the circumference of the patient's upper arm and the cuff width should be 40% of the length of upper arm. The centre of the bladder should be placed over the brachial arterial pulse. If these criteria are not met, the pressure generated by the air bladder may not be correctly transmitted to the brachial artery. A bladder that is too short can lead to overestimation of BP, up to 50 mmHg in obese patients, while bladders that are too wide cause readings that are too low.

ARM CIRCUMFERENCE	CUFF NAME	CUFF SIZE
22-26 cm	Small adult	12*22 cm
27-32 cm	Adult	16*30 cm
35-44 cm	Large adult	16*36 cm
45-52 cm	Adult thigh	16*42 cm

Technique for blood pressure measurements:

The technique used to measure BP is important in order to ensure accurate detection and diagnosis of hypertension, which is especially true during the initial visit by the patient to the clinic. Whether the BP differs between the two arms is important because in just over one-third of patients the systolic pressure may differ by at least 10 mmHg. The arm that displays the higher pressure should be used for further measurements. BP may also vary with postural changes. Generally, the systolic pressure will decrease and the

diastolic pressure will increase a few mmHg as a patient stands. Elderly people can be overly sensitive to standing, with 10% of those over the age of 65 years having a drop of 20 mmHg upon standing (4). BP should be taken after sitting quietly for five minutes, immediately after standing, and two minutes after standing. If these measurements differ by only a few mmHg, BPs may be taken only while the patient is seated during follow-up examinations. Also ascertain if a patient has recently (within one hour) exercised or consumed nicotine, caffeine, or alcohol, as all of these substances can affect BP. Patients should also be relaxed before measuring BP, since stress or activity can cause higher BPs. The patient should be seated for five minutes before measurement and should not speak or move during the measurement. The BP cuff should be placed around the upper arm such that the edge of the cuff is 2.5 cm (1 in) from the antecubital space. It does not matter if this is over the bare arm or over a single shirt sleeve. The cuff should be at heart level. If the arm is allowed to hang, the cuff could be as much as 15 cm (6 in) below the level of the heart, which could increase the BP by 10–15 mmHg because of hydrostatic pressure due to gravity (5). The stethoscope should not be used in such a way that it applies excess pressure to the arm. The bell of the stethoscope is recommended rather than the diaphragm, which could increase the delay in hearing the pulsation and result in underestimation of diastolic pressure by as much as 15 mmHg. The cuff should be inflated to a pressure approximately 30 mmHg above the estimated systolic pressure and then deflated at a rate of 2–3 mmHg per heartbeat. The systolic pressure occurs when the pulse can be felt in the

brachial artery as the blood re-enters the artery. Using auscultation, this is the point where the pulse is first heard and is termed the Korotkoff phase 1 sound. The pulse will continue to be heard as the cuff is deflated. At a pressure of 8–10 mmHg above the diastolic pressure, the sound often becomes muffled (Korotkoff phase IV) and then pulse sounds will cease (Korotkoff phase V). The diastolic pressure is recorded as the point at which the pulse sounds end, unless the spread between Korotkoff phases IV and V is more than 10 mmHg, in which case the phase IV point is recorded as the diastolic pressure (6).

Steps in measurement of blood pressure:

Place the stethoscope gently over the brachial artery at the point of maximal pulsation. Hold stethoscope firmly and evenly but without excessive pressure because excess pressure which may distort artery and produce sounds below diastolic pressure. Stethoscope end-piece should not touch clothing, cuff, or rubber tubes to avoid friction sounds. Inflate cuff rapidly to about 30 mm Hg above systolic pressure, deflate cuff at a rate of 2-3 mm Hg per pulse beat during which Korotkoff sounds will be heard. Deflate cuff rapidly after all sounds disappear. Make sure cuff is completely deflated before repeating measurement so as to avoid venous congestion of the arm.

Determinants of blood pressure:

The BP is determined by cardiac output, total body fluid volume, and resistance of blood moving through the arterial system. Also many environmental factors alter the function of organ systems, thereby generating a

complex interaction of multiple factors leading to difficulty in diagnosis of cause of primary hypertension. Cardiac output is the amount of blood pumped by the heart during each cardiac cycle which is determined by stroke volume and heart rate (7). Left ventricular stroke volume is the difference between the end diastolic volume (EDV) and end systolic volume (ESV). Stroke volume is dependent on the preload, the afterload, and the contractility of the heart.

Preload is the amount of stretch of the cardiac muscle due to blood filling the heart, which is controlled by the EDV filled by venous return from circulation. Any factor that alters the returning volume will alter the EDV and the preload. Changes in the EDV affect the stroke volume and thereby the cardiac output. A more rapid heart rate, for example, diminishes the amount of blood filling the ventricles and thus decreases preload. Afterload is back pressure from the arteries near the heart because of blood movement. Afterload is relatively constant and does not normally make a large contribution to changes in stroke volume. In patients with hypertension, however, afterload is more important, as elevated BP reduces the amount of blood ejected from the heart with each heart contraction (8). This leaves more blood in the heart after each contraction, raising the ESV and thus reducing the stroke volume. An extrinsic factor affecting stroke volume is cardiac contractility. Cardiac contractility is termed extrinsic because it does not depend on myocardial stretch. Increased contractility increases the force and amount of blood ejected with each heartbeat. This reduces the ESV, increasing the stroke volume.

Contractility is increased due to cytosolic increase in calcium ions prior to contraction.

The components that regulate cardiac output rely on the baroreceptors that act as mechanoreceptors that sense changes in vessel wall stretch and produce a rapid response to alterations in BP (9). They act as a buffering system to moderate normal short-term changes in BP. In hypertension, the increased stretching of arterial walls activates these receptors, causing inhibition of the vasomotor centre and resultant reduction in heart rate (to lower CO) and vasodilation (to decrease SVR). These changes reduce the BP. If the BP is too low, these receptors sense the reduction in arterial pressure and increase the CO and stimulate vasoconstriction. Their effect on long term blood pressure control is not known. Baroreceptors may become less sensitive with sustained elevation in blood pressure seen in hypertension. Blood volume is also an important factor that determines blood pressure. Blood volume and blood pressure are directly proportional to each other. As blood volume rises, blood pressure also rises. Blood volume influences venous pressure and ventricular filling, which alters the EDV and stroke volume. The kidneys have an important role in controlling blood and fluid volume. They can directly alter blood volume by increasing the filtration of fluid and reducing sodium retention and the fluid that accompanies it. Indirectly, the kidneys can regulate the renin–angiotensin–aldosterone system (RAAS) to change BP and ultimately increase the blood volume (10).

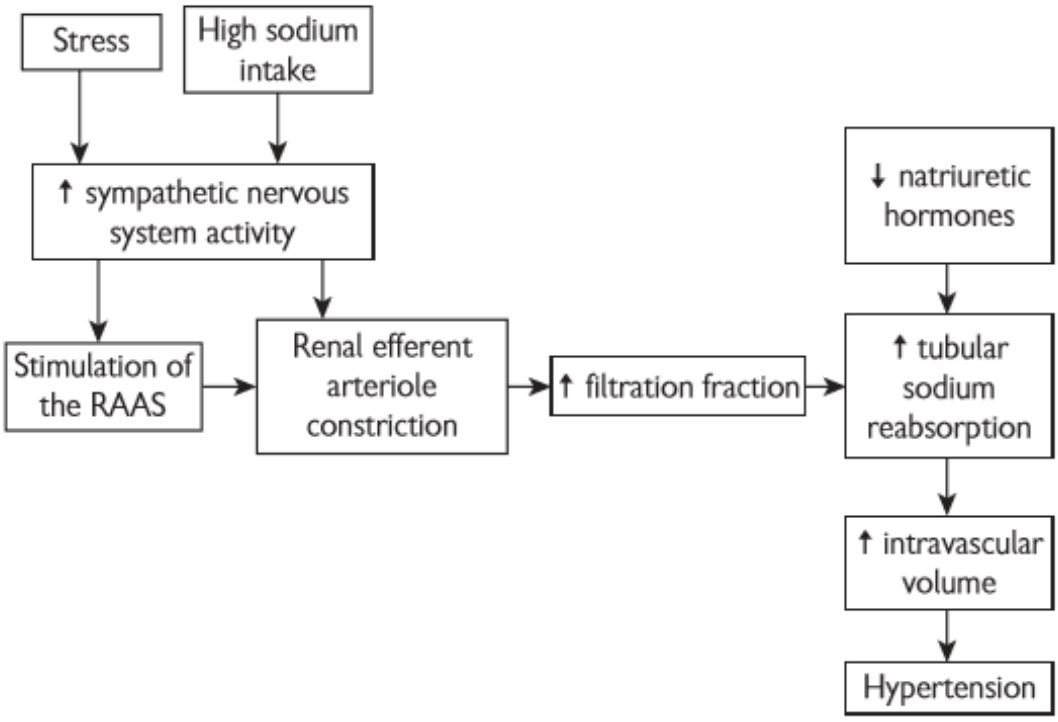
Regulation of blood pressure:

Systemic vascular resistance (SVR): SVR is the resistance to blood flow in the arterial tree. Arteries are composed of endothelial cells, vascular smooth muscle cells, and connective tissue. The intrinsic myogenic tone of the vascular smooth muscle and the sympathetic nervous system (SNS) control the diameter of the vessel, which influences the SVR. The nervous system and the endothelial cells play a major role in modifying smooth muscle cell tone. There are several vasodilators that reduce SVR, notably nitric oxide. Nitric oxide activates guanylatecyclase in smooth muscle cells, resulting in vasodilatation. Constriction of smooth muscle cells increases SVR. This system helps to maintain BP during stress, activity and during BP fall. Principal vasoconstrictors are angiotensin (AT) II (produced in response to release of renin by the kidney) and endothelin-1 (ET-1) produced by endothelial cells. ET-1 binds to receptors of vascular smooth muscle cells to activate voltage-dependent calcium channels. AT II binds to the G-protein coupled receptor AT1 to increase cytoplasmic calcium concentrations.

Systemic and local hormones, metabolites, and neurotransmitters all contribute to signalling pathways that affect CO and SVR. The neurotransmitters and hormonal signalling molecules involved in regulation of BP include Norepinephrine/ epinephrine, Angiotensin II, Endothelin, Nitric oxide, and Atrial natriuretic peptide/brain natriuretic peptide, Acetylcholine, Prostaglandins, Aldosterone, Bradykinin and Vasopressin (11). The parasympathetic nervous system mostly functions to down regulate the system

and the main parasympathetic neurotransmitter, acetylcholine reduces the heart rate. The sympathetic nervous system counteracts the relaxed state that dominates under parasympathetic activity to prepare the body for activity or stress through its main neurotransmitter, epinephrine. It acts through adrenergic receptors using the neurotransmitters epinephrine and norepinephrine of which the subclasses alpha2, and beta1 adrenergic receptors are most important in increasing and regulating BP. Stimulation of postsynaptic alpha1 and alpha2 adrenergic receptors located on smooth muscle cells causes vasoconstriction of the vessels. Activation of beta1 adrenergic receptors in heart muscle causes increased heart rate and increases the amount of calcium in the muscle cells, thereby increasing cardiac contractility. Cardiac output is raised due to increased stroke volume and heart rate. On stimulation of Beta1 adrenergic receptors within the kidney renin is released which results in the production of the vasoconstrictor AT II which thereby increases the blood pressure. Activation of renin angiotensin aldosterone system (RAAS) in excess amount can cause pathological consequences like sodium retention, endothelial dysfunction, inflammation and sympathetic activation. RAAS is a hormonal system that works to regulate BP and fluid volume through several mechanisms (12). Decrease in circulating blood volume causes renin release which causes conversion of angiotensinogen to Angiotensin 1 which in turn is converted to angiotensin II by angiotensin converting enzyme(ACE). Angiotensin 2 is an active molecule that stimulates two subtypes of receptors. Stimulation of the AT1 receptor subclass causes increase in BP through several mechanisms. The

first mechanism to increase blood fluid volume is through action on the proximal renal tubule causing sodium reabsorption. AT1 receptor activation also stimulates aldosterone release, causing further salt retention by the kidney. Thus all mechanisms of angiotensin 2 and stimulation of angiotensin 1 receptors lead to increase in blood pressure.



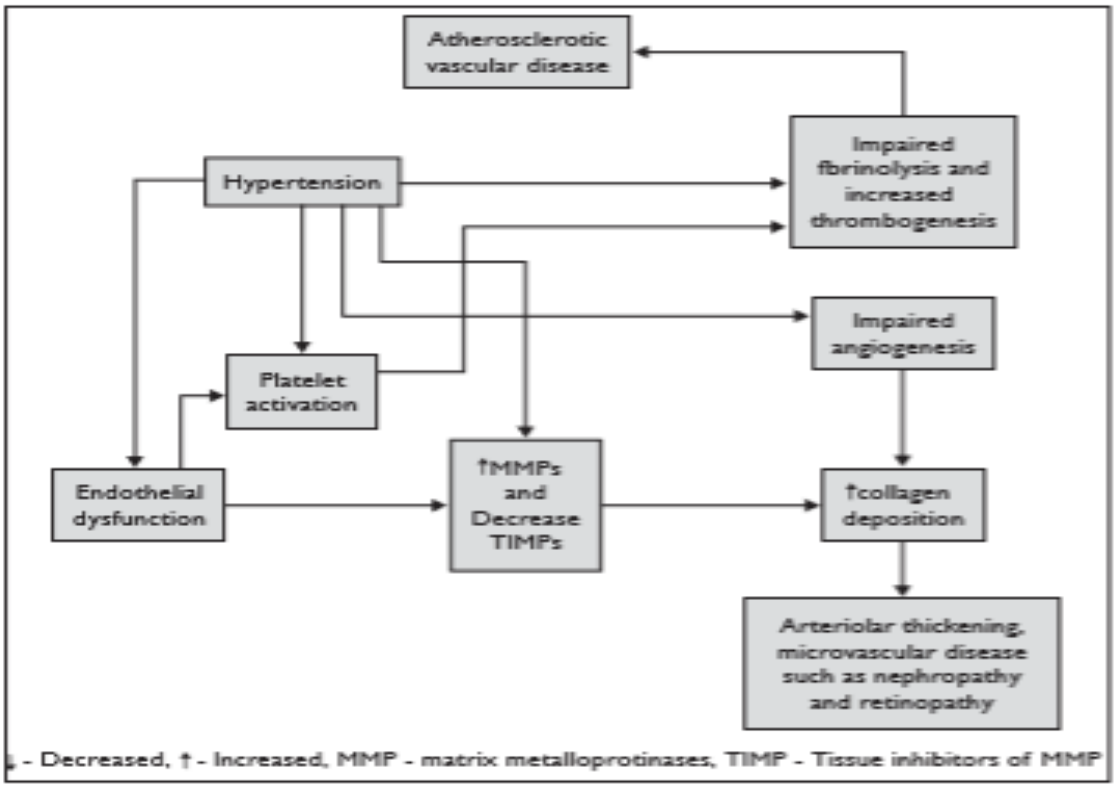
Risk factors for primary hypertension and its regulation:

Although the cause of primary hypertension is unknown, there are many risk factors associated with hypertension. Children with hypertensive parents are more than twice as likely to develop hypertension, suggesting a genetic component. Epidemiological evidence also suggests that up to 30% of BP varies due to genetic components (13). Hypertension as a whole is more severe in the black population compared with other races. Also factors associated with

lifestyle and environment like increased sodium intake, increased body mass, increased alcohol intake, increased psychological stress, increased physical activity, genetic predisposition, reduced potassium intake and reduced calcium intake may contribute to an increased risk of developing hypertension. Diet can enhance the susceptibility to develop, and once developed, sustain hypertension. Excess sodium intake and alcohol have both been linked to an increased likelihood of hypertension (14).

Obesity in the elderly is the main risk factor associated with hypertension in association with psychological stress and lack of physical activity. Also the contribution of each risk factor depends on individual patient susceptibility.

Being overweight also puts a person at increased risk of developing other risk factors associated with hypertension, including CVD and LVH. It also increases low-density lipoprotein cholesterol, lowers highdensity lipoprotein cholesterol, reduces glucose tolerance, and increases insulin resistance, all of which contribute to an increased risk of high BP. Activity of the RAAS is increased in overweight persons, leading to vasoconstriction and increased SVR. Obesity also causes alterations in insulin resistance, glucose tolerance, and dyslipidaemia all contributing to elevated BP. Finally, obesity can increase the frequency of obstructive sleep apnoea, a condition associated with secondary hypertension.



Sodium balance correlates with raised BP. Primary hypertension is more common in populations that consume higher amounts of sodium, especially if the average sodium intake is 100 mEq/day or more, but it is rare in populations that consume less than an average of 50 mEq/day (15). Reducing sodium intake can have a beneficial effect on BP. Sodium reduction from 170–100 mEq/day can reduce systolic BP by 5 mmHg and diastolic BP by 3 mmHg on average. JNC 7 recommends that all persons with a sodium intake of 100–150 mEq/day should reduce their sodium intake to less than 100 mEq/day. Changes in BP due to excess sodium intake reflect sodium sensitivity. Sodium sensitivity varies among individuals, and it increases with age or obesity. Non-Hispanic blacks are also more susceptible to sodium sensitivity and individuals with renal dysfunction have greater sodium sensitivity than people with normal

kidney function. The relationship between sodium and BP is not fully understood, but it may be related to fluid volume. If the level of sodium intake overwhelms the ability of the kidneys to filter sodium, sodium retention occurs which contributes directly to excess fluid volume and thereby hypertension. Sodium causes activation of signalling pathways leading to inappropriate vasodilation or vasoconstriction. It also exacerbates other risk factors such as micro albuminuria and dyslipidaemia.

Many genes have been linked to hypertension. One example of a single gene mutation leading to hypertension is Liddle's syndrome, caused by the dominant gain of function mutation in the sodium channel that prevents the degradation of the channel and leads to channel over activity causing excessive sodium reabsorption and systemic volume overload leading to early and severe hypertension. Many genes have been associated with primary hypertension but the individual contributions of each gene are currently unknown. There is evidence that genes active in the kidney are the major contributors to genetic-based hypertension. There is evidence that the AT, adducin, and connexin 40 genes have a pathogenetic role in high BP (16).

Physiological stress leads to activation of the sympathetic nervous system and can lead to vasoconstriction and changes to SVR. White coat hypertension which is stress of being in clinics is one of the leading causes of pseudo resistant hypertension. In the long term, stress can lead to sustained elevations of blood pressure. Lack of physical activity can contribute to essential hypertension by leading to higher stress levels, greater risk of obesity,

and reduced cardiovascular function. Studies have shown that reduced physical activity leads to increased body mass which contributes to hypertension.

Secondary hypertension: Recognition of secondary hypertension is important because hypertension is corrected by treatment of the underlying condition, which includes obstructive sleep apnoea, renal artery stenosis, chronic kidney disease, pheochromocytoma, Cushing's disease, thyroid disorders, hyperparathyroidism, brainstem compression, Medications, Pregnancy.

When a secondary cause is detected, it can be treated directly, often leading to improvement in BP. Renal disease hypertension is a major cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD). Patients with acute kidney injury (AKI) and CKD may become hypertensive due to renal dysfunction. The close association between renal function and BP is because the kidney is responsible for fluid filtration and salt regulation, both of which have direct impact on circulating fluid volume. Studies have shown that the prevalence of hypertension is inversely correlated with the glomerular filtration rate (GFR). Primary aldosteronismis elevated production of aldosterone leading to large increases in sodium and water retention. Patients with primary aldosteronism (PA) also have elevated BP due to changes in fluid handling.is result suggests that spironolactone may be useful for patients with essential hypertension even when PA is not present.

Pheochromocytomas are tumoursof adrenal medulla that secrete very large amounts of catecholamines leading to changes in SVR and CO and an increase in BP. It can also occur in other locations within the sympathetic chain. Pheochromocytoma is a relatively rare cause of secondary hypertension accounting for less than 1% of all cases of secondary hypertension.Approximately 85–95% of patients with a pheochromocytoma have hypertension, which may be either sustained or paroxysmal (19).

Obstructive sleep apnoea is also a cause of hypertension. It is characterized by multiple episodes of apnoea per night, in some instances as many as five or more airway obstructions per hour. The mechanism leading to rise in BP is obstruction in airflow resulting in asphyxia and lack of oxygen leading to hypoxia. Heart rate and stroke volume both change in response to hypoxia, which leads to changes in CO. Sleep apnoea can cause very large increases in short-term BP levels; systolic pressures immediately following an episode may be as high as 300 mmHg (20). One of the most efficacious treatments for obstructive sleep apnoea is continuous positive airway pressure breathing.

Cushing’s disease is caused by overproduction of cortisol. At high concentrations, cortisol can exhibit strong mineralocorticoid signalling that leads to hypertension. Interestingly, the morbidity and mortality associated with Cushing’s disease is most often due to diastolic hypertension. Other types of endocrine deregulation, such as disorders of the thyroid, hyperparathyroidism, and acromegaly, are also associated with hypertension.

Females may become hypertensive when taking oral contraceptives. Changing to other formulations of medication or other methods of birth control may improve BP control. Finally, excessive alcohol intake may lead to hypertension, with people consuming two or more alcoholic drinks per day having a two-fold risk of developing hypertension. Decreased alcoholic intake can lead to BP reduction. Many over the counter drugs can also cause hypertension.

The main goal of treating hypertension is to reduce its complications. The prevention and detection of hypertension related end organ damage is important in the stratification of cardiovascular risk for the patient. The assessment of target organ damage provides important information on the severity of the hypertension. It is also vital for management and prognostication. As hypertension prevalence increases with increasing age, early methods for prevention and detection of hypertension is necessary. Organ damage regresses with adequate therapy when done at a reversible stage. Several studies have found that hypertensive end organ damage and its modification with treatmentcorrelate more closely with ambulatory 24-hour blood pressure measurement than with office based blood pressure readings.

Factors which influence blood pressure in hypertension include pressure load, sympathetic nervous system, and renin angiotensin system, metabolic and inflammatory factors. Obesity, diabetes mellitus, high salt diet, dyslipidaemia all have influence on severity of hypertensive end organ damage (21). This indicates that blood pressure alone cannot be an independent factor for

predicting end organ damage also reduction of non-hemodynamic factors result in reduction in variable amounts of early hypertensive end organ damage which depends on the mechanism of action of antihypertensive agent used.

Routine biochemistry for renal function tests is useful prior to therapy, to monitor the effects of therapy and to rule out secondary causes. A decrease in potassium with or without an increase in sodium and increase in bicarbonate in the context of a patient not on diuretic therapy must alert the possibility of an adrenal cause of hypertension or secondaryhyperaldosteronism

Basic clues to secondary hypertension:

Decreased potassium	primary and secondary hyperaldosteronism Cushing's syndrome Liddle's syndrome Renal artery stenosis/disease
Increased potassium	Exclude artefacts (e.g. delayed analyses) Renal artery stenosis/ disease unmasked by ACEI/ARB Renal failure Immunosuppression Thrombocythaemia
Decreased sodium	Diuretics Syndrome of inappropriate antidiuretic hormone (SIADH) Intracranial pathology Hypothyroidism Hyperparathyroidism

Increased sodium	Dehydration Primary and secondary hyperaldosteronism
Increased creatinine	After commencement or dose escalation of ACEI or ARA should prompt investigation of RAS as an underlying cause At the time of presentation with malignant hypertension indicates a poorer prognosis especially if the serum creatinine more than or equal to 3mg%

Suspicion of secondary hypertension and its early diagnosis prevents the early occurrence of complications and prompt control of blood pressure.

Diagnosis of early hypertensive end organ damage (22):

- Left ventricular hypertrophy

  ECG: Sokolow-Lyon ≥ 38 mm, Cornell QRS > 244 mV*msec) or

  LVH(≥ 125 g/m2 for men and ≥ 110 g/m2 for women)

- Ultrasound examination for arterial wall thickening, (intima-media thickness [IMT] > 0.9 mm or arterio sclerotic plaque)

- Pulse wave velocity > 10 to 12 m/sec, depending on the device used

- Ankle-Brachial Index < 0.9

- Serum creatinine elevated - Men 1.3–1.5 mg/dL (115–133 µmol/L) ; Women 1.2–1.4 mg/dL (107–124 µmol/L)

- Elevated albumin excretion -Micro albuminuria 30–300 mg/24 hours; Albumin-creatinine ratio: men ≥ 22, women ≥ 31 mg/g creatinine
- Calculated glomerular filtration rate (<60 mL/ min/1.73 m2) or creatinine clearance <60 mL/min

Cardiovascular diseases and blood pressure:

Stroke is one of the most devastating consequences of hypertension and results in premature death or considerable disability. About 80% of strokes in patients with hypertension are ischaemic, being caused by an intra-arterial thrombosis or embolization from the heart or carotid arteries. The remaining 20% of cases are the result of various haemorrhagic causes. Amongst treated hypertensive patients, the risk of stroke is closely related to the accuracy of blood pressure control. Hypertension is also associated with an increased risk of atrial fibrillation. The presence of both conditions is additive to the risk of stroke. The incidence of stroke in patients with both conditions is 8% per year.Hypertension is a featured risk factor in stroke risk assessment scores for atrial fibrillation, such as the CHA2 DS2– VASc scores (23). Uncontrolled blood pressure substantially increases the risk of stroke in atrial fibrillation, even amongst anticoagulated patients.

PARAMETER	ALPHABET	SCORE
Congestive cardiac failure or left ventricular dysfunction	C	1
Hypertension ≥140/90 mm Hg	H	1
Age ≥75 years	A	2
Diabetes mellitus	D	1
Stroke / TIA /systemic thromboembolism	S	2
Vascular cause(previous myocardial infarction, PVD, or aortic plaque)	V	1
Age 65-74 years	A	1
Female	Sc(Sex category)	1
Maximum possible score		9

Hypertensives with hypokalaemia, due to diuretics or to aldosterone excess, are particularly at risk of developing atrial fibrillation and other arrhythmias. Abundant evidence from clinical trials shows that lowering blood pressure prevents all kinds of stroke. Recent evidence suggests that the β blockers are less effective at preventing stroke than other antihypertensive agents.Elderly people with hypertension are at risk of all forms of stroke and frequently sustain multiple small, asymptomatic cerebral infarcts that may lead to progressive loss of intellectual or cognitive function and dementia. An association also exists between hypertension and Alzheimer's disease. Hypertension is associated with increased risk of vascular dementia. But blood

pressure lowering in later life does not prevent the development of dementia or cognitive impairment in hypertensive patients with no apparent prior cerebrovascular disease (24). Coronary artery disease was more common than fatal stroke. Adequate treatment of hypertension reduces the risk of heart attack by about 20%. Many drugs used for the acute coronary syndromes and hypertension commonly treat both these conditions simultaneously. Hypertension may lead to coronary heart disease because of its contribution to the formation of coronary atheroma, with an interaction with other risk factors such as dyslipidaemia and diabetes mellitus.Left ventricular hypertrophy (LVH) is a common manifestation of hypertensive target organ damage. Eccentric LVH was more common than concentric hypertrophy. LVH occurs as a result of increased after load on the heart, caused by raised peripheral vascular resistance. Subsequently, the increased muscle mass outstrips its blood supply and this, coupled with the decreased coronary vascular reserve, can result in myocardial ischemia – even in patients with normal coronary arteries. High intake of salt and increased levels of angiotensin II in the plasma increase the chances of developing LVH. The angiotensin blocking drugs reduce LVH more than other classes of drug. The prevalence of LVH is similar in patients with isolated systolic hypertension and systolic–diastolic hypertension (25). LVH secondary to hypertension is a major risk factor for myocardial infarction, stroke, sudden death and congestive cardiac failure. This increased risk is in addition to that imposed by hypertension itself.In addition, patients with hypertension and LVH are at increased risk of cardiac arrhythmias (atrial

fibrillation and ventricular arrhythmias) and atherosclerotic vascular disease (coronary and peripheral artery disease). When LVH on the ECG is accompanied by repolarisation abnormalities it is also called ‘strain’ pattern which is associated with higher morbidity and mortality. Epidemiological studies like Framingham Heart Study have shown that hypertension is the principal cause of heart failure. People with blood pressure >160/95 mm Hg have a six fold higher incidence of heart failure than those with pressures <140/90 mm Hg. Hypertension as a cause of heart failure is confounded by the underlying predisposition to coronary artery disease. Most cases of heart failure are the result of left ventricular systolic dysfunction that results from damage to the ventricle after myocardial infarction. The presence of LVH on an electrocardiogram itself significantly increases the risk of heart failure. The presence of gross LVH can result in impaired ventricular compliance and relaxation, which leads to diastolic heart failure. This leads to Heart failure with preserved ejection fraction. This results in left atrial dilatation and precipitation of atrial fibrillation. The development of atrial fibrillation per se can precipitate pulmonary oedema, especially if LVH and diastolic dysfunction are present.

Hypertension has traditionally been associated with heart failure, and it has been relatively easy to infer empirically a cause and effect relationship. However, although the evidence is irrefutable that hypertension is a risk factor for heart failure it has been less clear that hypertension is a causal factor for heart failure. Also it is important to recognize the unique contribution of

hypertension to HFpEF, a phenotype of heart failure which is now the predominant clinical syndrome recognized in hospital settings and responsible for more than 50% of all acute heart failure admissions (26). Unlike HFrEF where clarity of the pathophysiology exists, the cellular and molecular aspects of the pathophysiology of HFpEF remain elusive. Prevailing considerations implicate fibrosis, ventricular noncompliance, hypertrophy, and ischemia the factors impacted by hypertension. Hypertension when aligned with coronary artery disease, obesity, diabetes and atrial fibrillation, explains the majority of concomitant comorbidities associated with clinical HFpEF (heart failure with preserved ejection fraction).

Hypertension in association with renal artery stenosis but with no intrinsic myocardial disease can cause ‘flash’ pulmonary oedema that is related to high levels of plasma renin and angiotensin. This can be corrected by treatment of the renal artery stenosis. Over many years, heart failure in association with untreated hypertension may lead slowly to a decrease in blood pressure as the left ventricular function progressively worsens. Patients whose hypertension mysteriously has normalised may have a bad outlook, as this normalisation is the result of a silent or clinically overt myocardial infarction or the development of left ventricular systolic dysfunction. Hypertension contributes to atheromatous vascular disease in all vascular beds. Peripheral artery disease manifested by intermittent claudication is about three times more common in patients with hypertension. These patients may also have renal artery stenosis, which may contribute to their hypertension (27). Disease in the

aorta coupled with hypertension may result in the development of abdominal aortic aneurysm. High pulsatile wave stress and atheromatous disease can lead to dissection of aortic aneurysms, which carries a high short-term mortality. Extra cranial carotid artery atheromatous disease is also more common in people with hypertension.Renal dysfunction commonly is associated with hypertension in the presence of diabetes and intrinsic renal disease. Whether mild-to-moderate essential hypertension alone leads to renal failure remains a controversy. Because hypertensive patients who develop progressive renal failure can have an undiagnosed primary renal disease. Malignant hypertension often leads to progressive renal failure. Almost all primary renal diseases cause an increase in blood pressure, which is mediated by high levels of renin andangiotensin, as well as sodium and water retention. There is increasing evidence of the prognostic importance of proteinuria, micro proteinuria and mild elevations of serum creatinine in patients with hypertension and no clear evidence of intrinsic renal disease. Patients with renal failure, with or without dialysis or transplantation, have a greatly increased risk of developing coronary heart disease or strokes. There is also marked excess of hypertension in patients following renal transplantation (28). Hypertension leads to vascular changes in the eye, which is referred to as hypertensive retinopathy, comprising of generalised and focal retinal arteriolar narrowing, arteriovenous nipping or nicking, retinal haemorrhages, micro aneurysms and, in severe cases, optic disc and macular oedema. These changes were classified by Keith, Wagener and Barker into four grades that correlate with prognosis. The most severe

hypertension – that is, malignant hypertension – is defined clinically as increased blood pressure in association with bilateral retinal flame shaped haemorrhages and cotton wool spots or hard exudates, or both, with or without papilledema. If hypertensive patients with malignant hypertension are left untreated 88% patients die within 2 years. Mild hypertensive retinopathy signs are seen in nearly 10% of the general adult non-diabetic population (29). Hypertensive retinopathy is closely associated with other indicators of end-organ damage (e.g. LVH, renal impairment) and may be a risk marker of future clinical events, such as stroke, congestive heart failure and cardiovascular mortality.Several retinal diseases such as retinal vascular occlusion (artery and vein occlusion), retinal arteriolar emboli, macro aneurysm, ischaemic optic neuropathy and age-related macular degeneration are also related to hypertension, although there is no evidence that treatment of hypertension prevents vision loss from these conditions.

Patients with hypertension are at increased risk of heart attacks, stroke and atrial fibrillation during general anaesthetics and the immediate post-operative period. In addition, quite marked surges in blood pressure are seen during the induction of anaesthesia and endotracheal intubation. In patients who are receiving treatment with drugs which block the renin–angiotensin–aldosterone system (the ACE inhibitors or the angiotensin receptor blockers), the height of the blood pressure is highly dependent on their intravascular volume and hydration status. Careful and accurate fluid replacement in mandatory. In patients with surgical emergencies who have very high blood

pressures, a diagnosis of phaeochromocytoma should be considered, although this is very rare. Emergency blood pressure reduction is best achieved either with intravenous nitrates or sodium nitroprusside infusion. Occasionally, oral nifedipine 30 mg can be used in hypertensive urgencies, but not in emergencies (30). Patients for non-emergency surgery with known and treated hypertension should continue their antihypertensive therapy until the morning of operation. Treatment should usually be restarted as soon as the patients are able to swallow their pills. Patients who undergo elective surgery will be very anxious and may develop raised blood pressures, not unlike the so-called white-coat effect. It is crucial therefore that the blood pressure is measured accurately in a quiet, conversation-free room with the patient seated, preferably using an automatic manometer. Up to five of six blood pressure readings should be taken at 5 min intervals. If the systolic blood pressure settles to below 160 mm Hg and there is absolutely no LVH or any other abnormality on the ECG, surgery can proceed as planned. If prior to non-urgent surgery the blood pressure remains above 160 mm Hg or the ECG shows LVH, the operation should be postponed till blood pressure control is achieved. Patients with hypertension who smoke cigarettes are particularly at high risk for surgery.

Monogenic hypertension should be considered secondary hypertension because an underlying genetic defect is clearly identifiable. The genetic defects that are necessary and sufficient for monogenic hypertension have distinctive characteristics that make them different from genetic variants underlying primary hypertension. Eight different monogenic hypertensive syndromes have

been described. Even collectively, monogenic familial hypertension is thought to be rare with an incidence of likely below 1/5000 in the general population (31). Even though likely rare, the genetic variants underlying MHS are important in because in some cases, specific treatment approaches exist which have spectacular treatment effects and because the recognition of the problem leads to early screening of family members and prevent complications. Secondly, identifying the cause can permit further research in the mechanisms underlying hypertension.

Elevated aldosterone	1.Glucocorticoid remediable aldosteronism 2.Gordon syndrome 3.Familial hyperaldosteronism type III
Low aldosterone	4.Liddle syndrome 5.Apparent mineralocorticoid excess
Low Aldosterone and Associated Features	6.Hypertension and brachydactyly syndrome (Bilginturan syndrome) 7.Autosomal dominant hypertension with exacerbation in pregnancy 8.Congenital adrenal hyperplasia type 4

Clinical Recognition of Monogenic Hypertension is through characteristics like early onset hypertension, young age, low renin levels, positive family history with significantly high blood pressure levels.

Hypertension and heart:Echocardiography has also played an essential role in elucidating the effects of high blood pressure on cardiac mechanical function leading to clinical heart failure. Although the direct link between hypertension and systolic dysfunction is often complicated by concomitant cardiovascular disease, there is a direct and stepwise relationship between LV mass and systolic dysfunction, independent of the prevalence of incident myocardial infarction. Diastolic dysfunction on the other hand referring to abnormalities in LV relaxation and filling, is a hallmark of hypertensive heart disease. This is due to LV remodelling that occurs in hypertension leading to cardiac myocytes hypertrophy, and fibrotic changes that increase LV stiffness and alter cardiac mechanical properties. Studies have shown that diastolic hypertension has 34% prevalence among elderly individuals. A newer technique called peckle-tracking echocardiography, which uses computer algorithms to track pixels of imaging data is a novel technique to directly measure and quantify myocardial displacement, velocity, and deformation (stretch or contraction). Abnormalities in these measures of cardiac mechanicals have been shown to be precursors of heart failure. Circumferential strain was associated with future heart failure risk in asymptomatic individuals, even after adjusting for age, diabetes, hypertension, myocardial infarction, LV mass, and LV ejection fraction. Blood pressure can adversely affect myocardial strain. This highlights that prevention of hypertension per se prevents more overt cardiovascular disease including heart failure.

Age-related haemodynamic patterns underlying hypertension:

Essential hypertension is characterized by derangements in 1 or more of the physiological determinants of BP. Age exerts a marked influence on which component becomes abnormal, and this corresponds closely to the form of essential hypertension which is observed. Adolescents and young adults (<30 years) with raised BP are often considered to have early, or borderline, hypertension. In this the principal haemodynamic disturbance is an increase in stroke volume, whereas peripheral vascular resistance (PVR) is relatively normal. In keeping with this physiological profile, isolated systolic hypertension is the predominant form of hypertension observed in young individuals (32). In contrast, in middle-aged individuals (~30–50 years), cardiac output is normal or even reduced, but the dominant haemodynamic disturbance is a markedly increased PVR, which is most likely due to structural remodelling of the resistance vasculature in response to continual exposure to higher pressures. Isolated diastolic hypertension (IDH) or mixed (systolic/diastolic) hypertension (SDH) are the predominant forms of hypertension observed in this age group. Systolic/ diastolic hypertension (mixed) is commonly viewed as the established or 'classical' form of essential hypertension. Individuals with age more than 50 years have arterial stiffness as the predominant hemodynamic disturbance that leads to isolated systolic hypertension. This causes an exaggerated increase in pulse pressure because the large arteries can no longereffectively buffer the cyclical changes in BP during each cardiac cycle. Hence the influence of age on diagnosis of

hypertension is essential because an elderly individual with diastolic hypertension is doubtful to be essential hypertension. Always secondary forms of hypertension is a possibility in these individuals.

Age	Principal hemodynamic disturbance	Predominant form of hypertension
<30 years	Increase in stroke volume	Isolated systolic hypertension
30–50 years	Increase in peripheral vascular resistance	Systolic/diastolic hypertension(mixed) or isolated diastolic hypertension
>50 years	Increase in arterial stiffness	Isolated systolic hypertension

Central versus peripheral blood pressure:

Moving from central (i.e. aorta) to peripheral (i.e. brachial) arteries, systolic pressure increases due to differences in vessel stiffness and wave reflections, whereas mean and diastolic pressure fall by only 1–2mmHg (33). This small fall in mean pressure causes blood to flow forwards, not backwards. The resultant widening or amplification of the pulse pressure— which is more pronounced in younger individuals—means that BP assessed at the brachial artery overestimates BP in the aorta and central arteries. The difference between brachial and central BP is important because it is the central pressure to which the heart, brain, and other major organs are exposed, and certain drug therapies exert differential effects on peripheral and central pressure. In addition, stratifying individuals by brachial pressure reveals considerable

overlap in aortic pressure which holds important implications for the future categorization of hypertension—if central BP is more important in defining an individual's risk and/or the impact of therapy, then categories that are based on central rather than peripheral pressure may be more useful.

Hypertensive retinopathy:

Hypertensive retinopathy is commonly seen in the eyes of patients with long-standing uncontrolled hypertension. These changes occur in the retina, optic nerve head, and choroidal circulation. The changes in the retina (hypertensive retinopathy) are the most widespread early changes that are seen and that have been described. There are many classifications for these changes, including the well-established Keith–Wagener–Barker classification and the Scheie classification. The Keith–Wagener–Barker classification was the first to correlate retinal findings with blood pressure while the Scheie classification was based on the fundoscopy findings alone. Nowadays these classifications do not correlate well with severity of hypertension and new simpler two-grade classification of non-malignant versus malignant retinopathy has been proposed. The most common ocular manifestation is narrowing of the retinal arterioles. In young patients, the arterioles may constrict due to auto regulation. In older patients, luminal fibrosis and vessel rigidity prevent the same degree of narrowing. At points where the retinal arteriole crosses over the retinal venule, compression of the vein may cause the appearance of arteriovenous nicking. Other changes include cotton wool spots (nerve layer micro-infarcts that obtain this appearance due to disruption of axoplasmic transport), dot/blot

hemorrhages, and flame-shaped haemorrhages. Under normal circumstances, there are many feedback mechanisms that maintain retinal flow despite changes in blood pressure. Retinal vessels have the ability to maintain a constant blood flow despite changes in perfusion pressures by either vasodilation or vasoconstriction. However, with hypertension, there is a breakdown of this mechanism, due to changes in endothelial-derived molecules. This breakdown of the auto regulation leads to other changes such as oedema and fibrosis. In malignant hypertension, the changes seen include papilledema as well as hard and soft exudates which are due to severe vasospasm of the vessels in response to the high pressures (as seen in malignant hypertension), leading to necrosis and focal leakage from the precapillary arterioles that lie deep in the retina.

The Keith–Wagener–Barker classification: (34)

Grading	Fundoscopic findings	Clinical correlates
Grade 1	Slight narrowing, sclerosis, and tortuosity of the retinal arterioles	Mild asymptomatic hypertension
Grade 2	Definite narrowing, focal constriction, sclerosis, and arteriovenous (AV) nicking	Blood pressure is higher and sustained, few if any symptoms attributable to high blood pressure

Grade 3	Cotton wool spots, haemorrhages	Blood pressure higher and more sustained, symptoms such as headaches, vertigo
Grade 4	As above, with papilledema, Elschnig spots	Persistently elevated blood pressure, headaches, visual disturbances, impaired cerebral and renal function

Modified Scheie classification of hypertensive retinopathy

Grade	Fundoscopic findings
0	No changes
1	Minimal arteriolar narrowing
2	Obvious arteriolar narrowing with focal irregularities
3	Grade 2 + retinal haemorrhages and/or exudate
4	Grade 3 + swollen optic nerve (malignant hypertension)

Hypertension and dementia:

The inverse association between blood pressure and cognitive impairment has been demonstrated in a number of epidemiological studies. The Framingham Heart Study (35) was one of the first to demonstrate that attention

and memory measures are inversely related to blood pressure levels and duration of hypertension. The mechanisms involved are not certain. Hypertension is a risk factor for atherosclerosis, stroke, or cerebral infarction, which lead to cognitive decline. In the absence of an overt cerebrovascular accident or stroke, cognitive impairment may be due to microvasculature occlusion.The Systolic hypertension in Europe Study was one of the first studies to demonstrate a protective effect of antihypertensive therapy on the development of cognitive impairment. Similar findings have been demonstrated in larger studies including the Perindopril Protection Against Recurrent Stroke Study (PROGRESS) and the Study on Cognition and Prognosis in the Elderly (SCOPE). Antihypertensive therapy has also been shown to reduce the incidence of white matter changes on magnetic resonance imaging.

Neurovascular Coupling

Neurovascular coupling links the metabolic demands of the neurons to cerebral perfusion. This requires rapid integrated signalling between neurons, interneurons, perivascular nerves, glia, and the cells in the vasculature. Active neurons, interneurons, and astrocytes release vasodilators that cause localized dilation, or functional hyperaemia, in penetrating arterioles and pial arteries supplying them. Neurovascular coupling requires the activity of the three key vasodilator pathways: nitric oxide (NO), cyclooxygenase (COX)-2 metabolites, and epoxyeicosatrienoic acids (EETs).

Effects of Hypertension on Neurovascular Coupling:

Studies of the effects of hypertension on neurovascular coupling in humans are limited. One study showed that increases in regional perfusion in response to a memory test were impaired in patients with untreated hypertension. It should be noted that the patients in this study were only mildly hypertensive (systolic/diastolic 144.2/84.4 mmHg). It is possible that patients with more malignant hypertension will exhibit more marked impairments in neurovascular coupling.

Hypertension and renal damage:

Renal disease has an important relationship with hypertension in that it could be either be a cause or an effect of hypertension. It is epidemiologically more common to see renal failure leading to hypertension, but the converse is controversial, except in malignant hypertension, where progressive deterioration of renal function has been demonstrated. The mechanisms involved here are similar to those seen for the retinal disease. Here as well, the glomerular vessels auto regulate the blood flow by vasoconstriction or vasodilatation depending on the perfusion pressures, to keep the actual perfusion at the glomerulus constant. Prolonged high perfusion pressures can lead to significant vasoconstriction, which can then cause localized damage to the glomeruli. This can cause necrosis of the glomeruli leading to micro albuminuria, which could lead to significant proteinuria if the disease is not treated. Renal failure in the absence of the malignant phase could also be an

effect of atherosclerosis affecting the renal arteries, leading to under perfusion. As with LVH, micro albuminuria has also been shown to correlate with future cardiovascular events. The reversal of micro albuminuria with the strict treatment of hypertension has been shown to improve cardiovascular events.

In hypertensive patients,simultaneous albuminuria/proteinuria and eGFR should be assessed to evaluate for end organ damage. Presence of micro albuminuria and eGFR less than 60ml/min are indirect evidence of increased cardiovascular risk. The gold standard for quantifying urine albumin is immunoassay using polyclonal sera. Dipstick analysis to look for albuminuria can be used as screening test.

An understanding of the different pathophysiological mechanisms involved in the causation of TOD in hypertension is important, as this would help us devise means of reducing catastrophic complications of hypertension. Whilst it has been shown convincingly that the use of antihypertensive agents reduces cardiovascular and cerebrovascular complications and that they reverse endothelial and platelet activation in hypertension, a direct correlation between the improvement in endothelial and platelet activation and a decrease in cardiovascular endpoints has not been shown. More studies are needed to fully understand the different mechanisms involved in the pathogenesis of target organ damage in hypertension and in devising strategies to prevent them.

LIST OF ABBREVATIONS:

NCD- Non communicable disease	ET-1 – Endothelin-1
PVR- peripheral vascular resistance	AT- angiotensin
SVR- systemic vascular resistance	CVD- cardiovascular disease
ESV- end systolic volume	LVH- left ventricular hypertrophy
EDV- end diastolic volume	IMT- intima medial thickness
CO- cardiac output	ESRD- end stage renal disease
SNS- sympathetic nervous system RAAS- renin angiotensin aldosterone system	HFpEF- heart failure with preserved ejection fraction
JNC- Joint National Committee	

MATERIALS AND METHODS

Source of study: data consists of primary data collected by the principal investigator directly from newly detected hypertensive patients attending NCD out-patient department in Coimbatore Medical College Hospital.

DESIGN OF STUDY: Cross sectional study

PERIOD OF STUDY: One year

It is a cross sectional study to assess the prevalence of target end organ damage in 200 newly detected hypertensive patients attending NCD OPD in Coimbatore Medical College Hospital, Coimbatore from June 2017 to June 2018.

Inclusion criteria:

Patients in the age group of 30 to 50 years attending NCD OPD newly detected as hypertensive patients

Exclusion criteria:

1) Known hypertensive patients
2) Age less than 30 years and more than 50 years
3) Patients with previous history of Coronary artery disease, cerebrovascular accident, diabetes mellitus, visual disturbances, renal failure, peripheral arterial occlusive disease

The data obtained were analyzed using SPSS version 21.0 software. Results were expressed in frequencies and percentages.

OBSERVATIONS AND RESULTS

TABLE 1-AGE DISTRUBUTION

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
< 40	31	15%
41-50	68	34%
51-60	56	28%
> 60	45	23%

CHART 1- AGE DISTRIBUTION

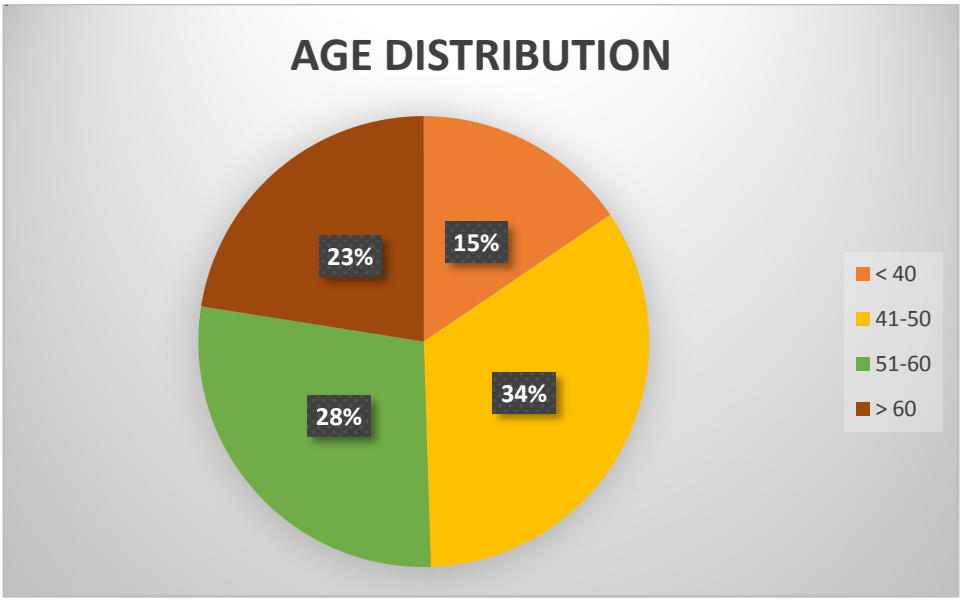


TABLE 2- SYSTOLIC BLOOD PRESSURE STAGES

SYSTOLIC BP	NO OF PATIENTS	PERCENTAGE
STAGE I HT	53	26%
STAGE II HT	147	74%

CHART 2 – SYSTOLIC BLOOD PRESSURE STAGES

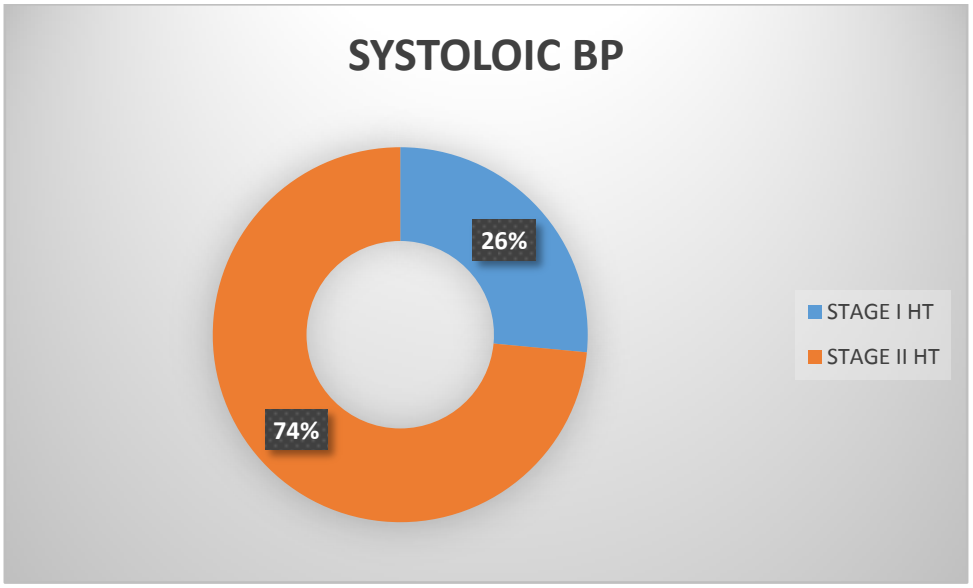


TABLE 3- DIASTOLIC BLOOD PRESSURE STAGES

DIASTOLIC BP	NO OF PATIENTS	PERCENTAGE
STAGE I HT	8	4%
STAGE II HT	192	96%

CHART 3- DIASTOLIC BLOOD PRESSURE STAGES

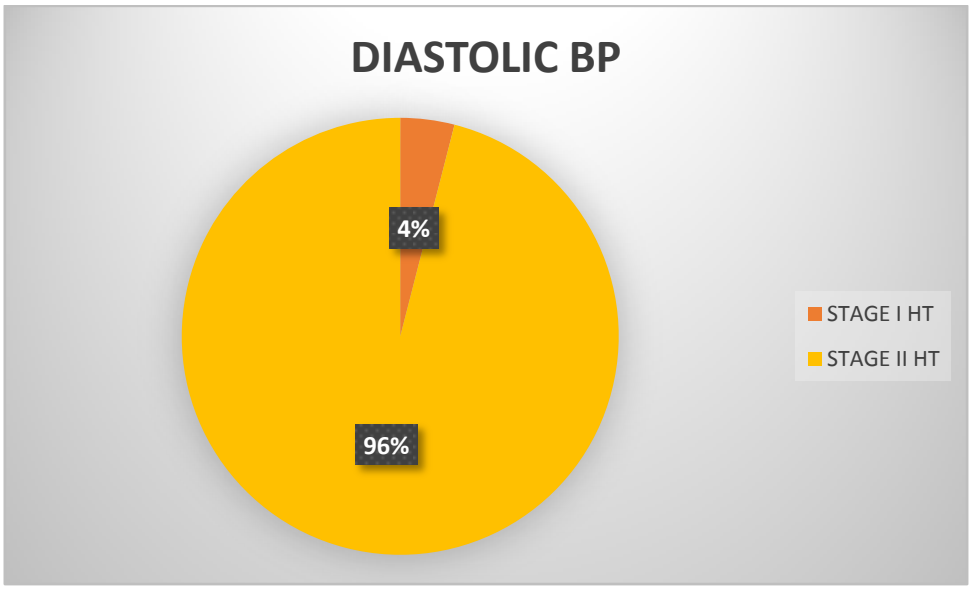


TABLE 4- SEX DISTRIBUTION

SEX	NO OF PATIENTS	PERCENTAGE
MALE	96	48%
FEMALE	104	52%

CHART 4- SEX DISTRIBUTION

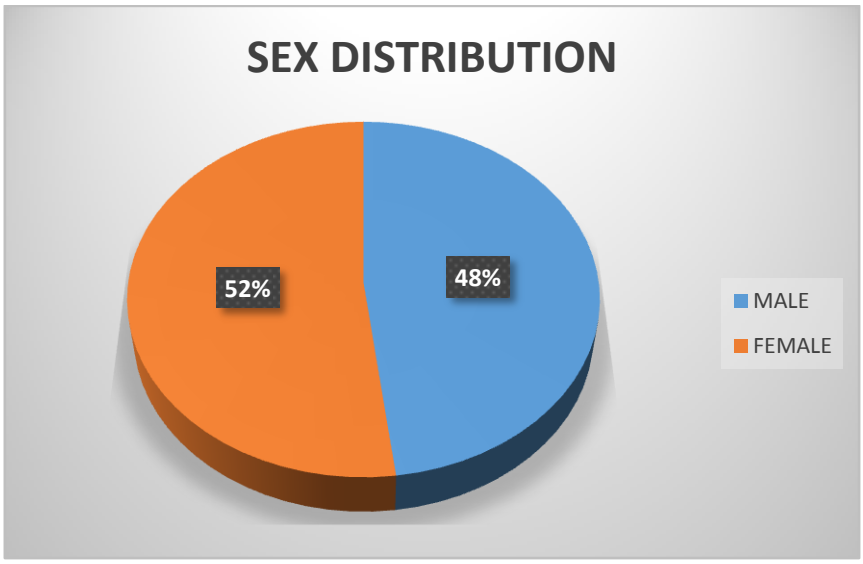


TABLE 5- COMPARISON OF STAGE OF SBP WITH SEX

SEX	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
MALE	26	70
FEMALE	27	77
CHI SQUARE TEST		
P VALUE - 0.857		
NON SIGNIFICANT		

CHART 5- COMPARISON OF STAGE OF SBP WITH SEX

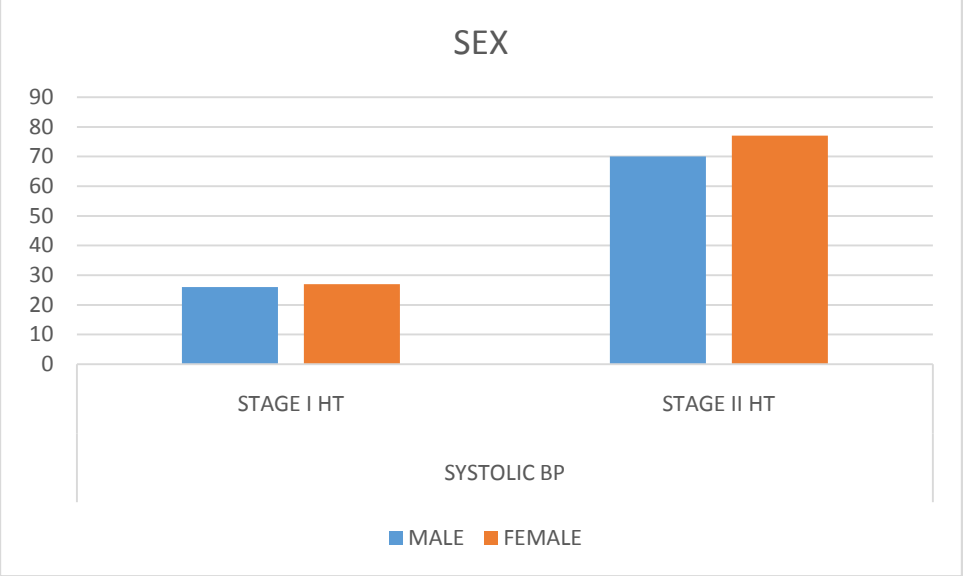


TABLE 6- COMPARISON OF STAGE OF DBP WITH SEX

SEX	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
MALE	4	92
FEMALE	4	104
CHI SQUARE TEST		
P VALUE - 0.408		
NON SIGNIFICANT		

CHART 6- COMPARISON OF STAGE OF DBP WITH SEX

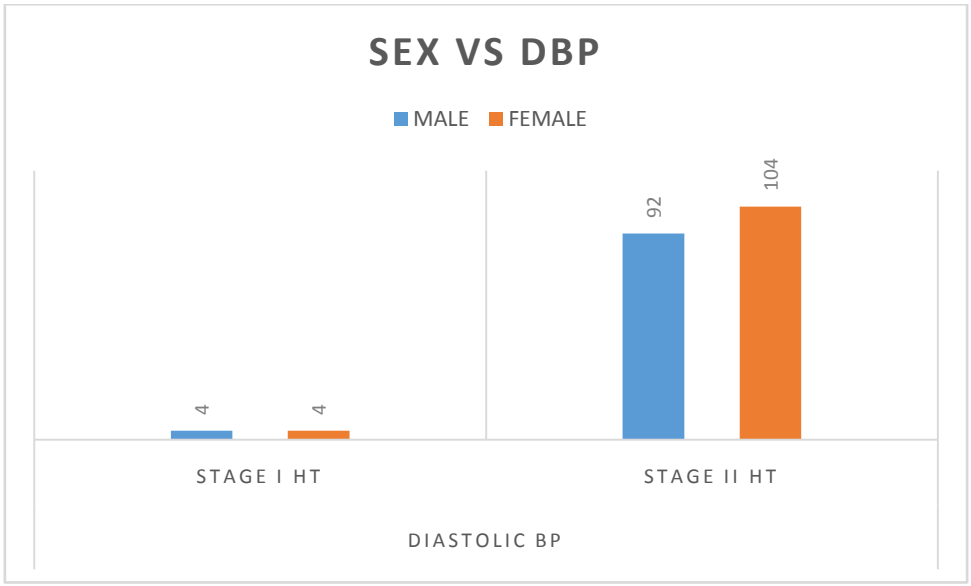


TABLE 7- COMPARISON OF MEAN SBP WITH SEX

SEX	SYSTOLIC BP	
	MEAN	SD
MALE	148.75	19.96
FEMALE	147.98	18.13
UNPAIRED T TEST		
P VALUE - 0.776		
NON SIGNIFICANT		

CHART 7- COMPARISON OF MEAN SBP WITH SEX

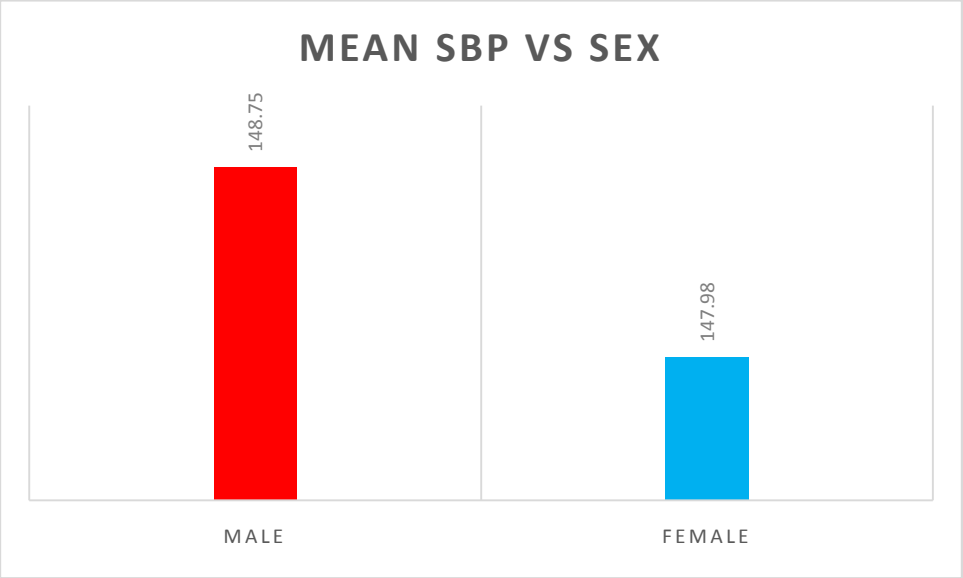


TABLE 8- COMPARISON OF MEAN DBP WITH SEX

SEX	DIASTOLIC BP	
	MEAN	SD
MALE	99.58	12.72
FEMALE	97.6	10.92
UNPAIRED T TEST		
P VALUE - 0.236		
NON SIGNIFICANT		

CHART 8- COMPARISON OF MEAN DBP WITH SEX

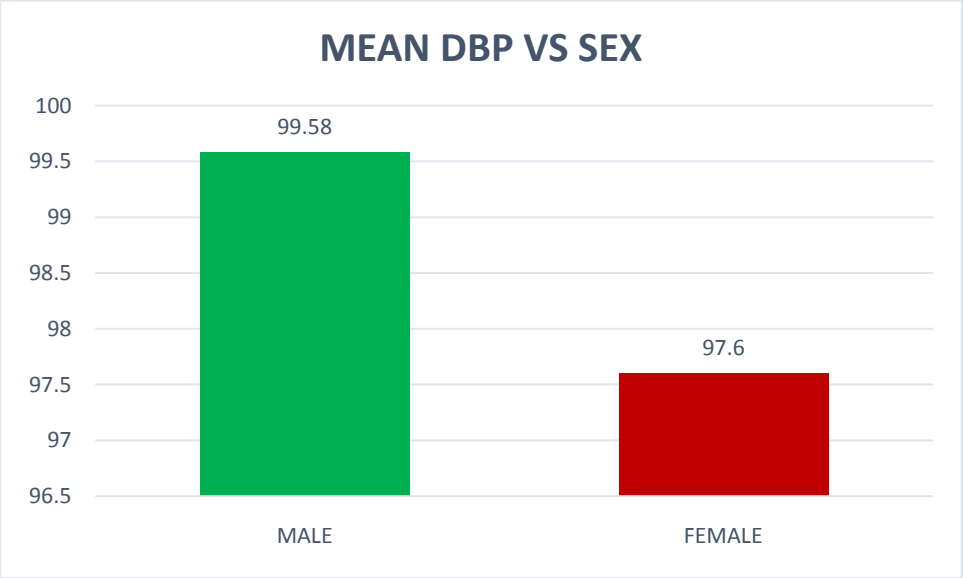


TABLE 9- BODY MASS INDEX DISTRIBUTION

BODY MASS INDEX	NO OF PATIENTS	PERCENTAGE
MORE THAN 25	97	48%
LESS THAN 25	103	52%

CHART 9- BMI DISTRIBUTION

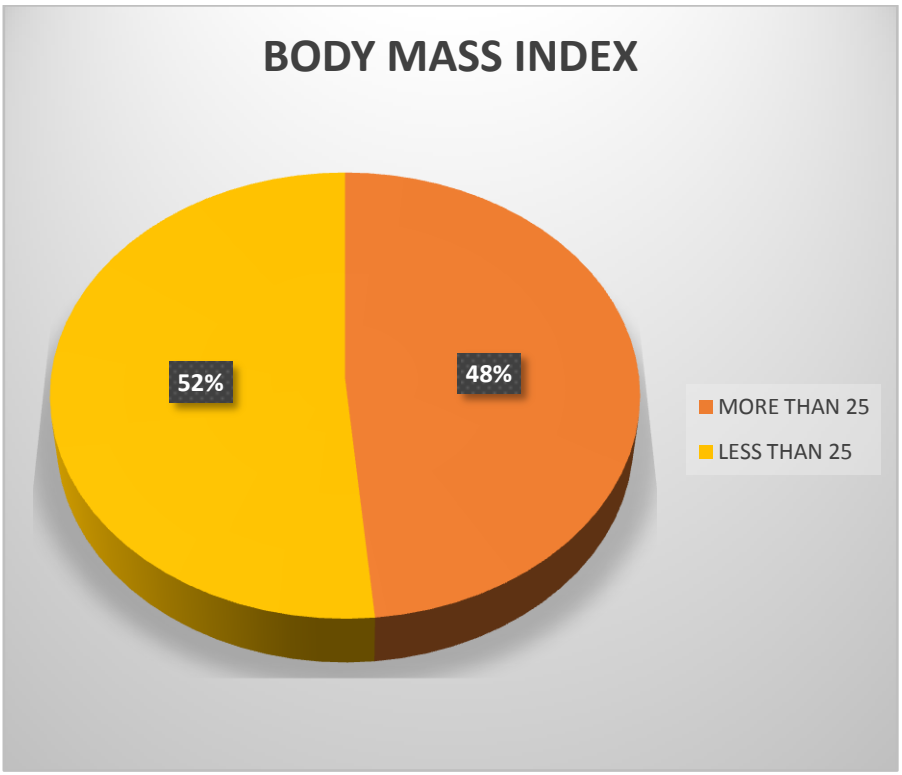


TABLE 10- COMPARISON OF STAGE OF SBP WITH MEAN BMI

SYSTOLIC BP	BMI	
	MEAN	SD
STAGE I HT	24.23	4.1
STAGE II HT	25.2	4.82
UNPAIRED T TEST		
P VALUE - 0.194		
NON SIGNIFICANT		

CHART 10- COMPARISON OF STAGE OF SBP WITH MEAN BMI

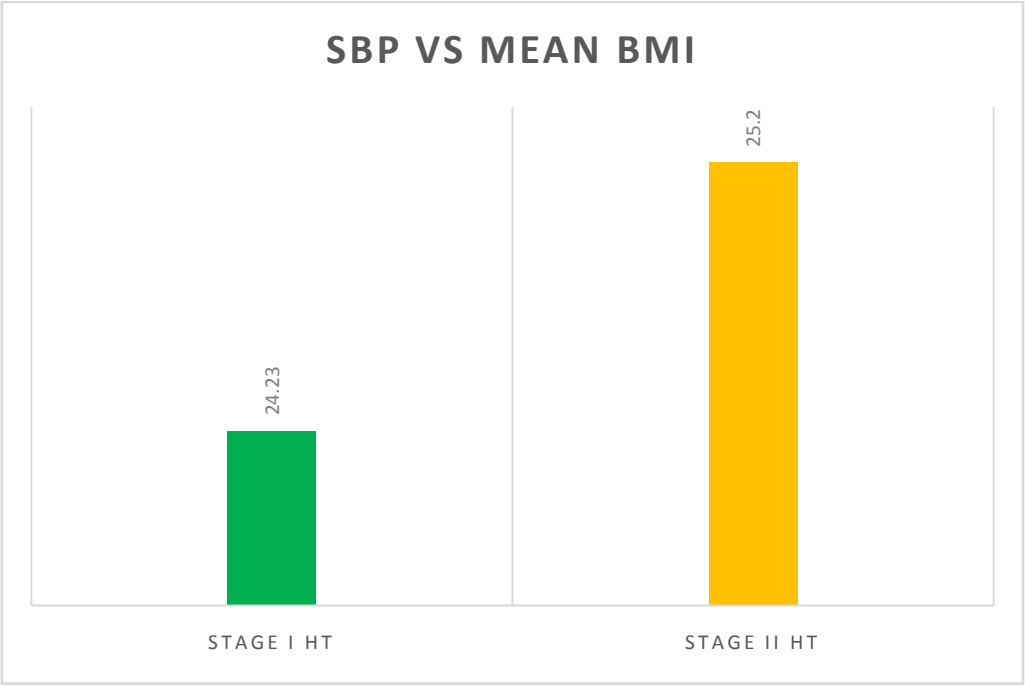


TABLE 11- COMPARISON OF STAGE OF DBP WITH MEAN BMI

DIASTOLIC BP	BMI	
	MEAN	SD
STAGE I HT	24.13	3.98
STAGE II HT	24.9	4.68
UNPAIRED T TEST		
P VALUE - 0.614		
NON SIGNIFICANT		

CHART 11-COMPARISON OF STAGE OF DBP WITH MEAN BMI

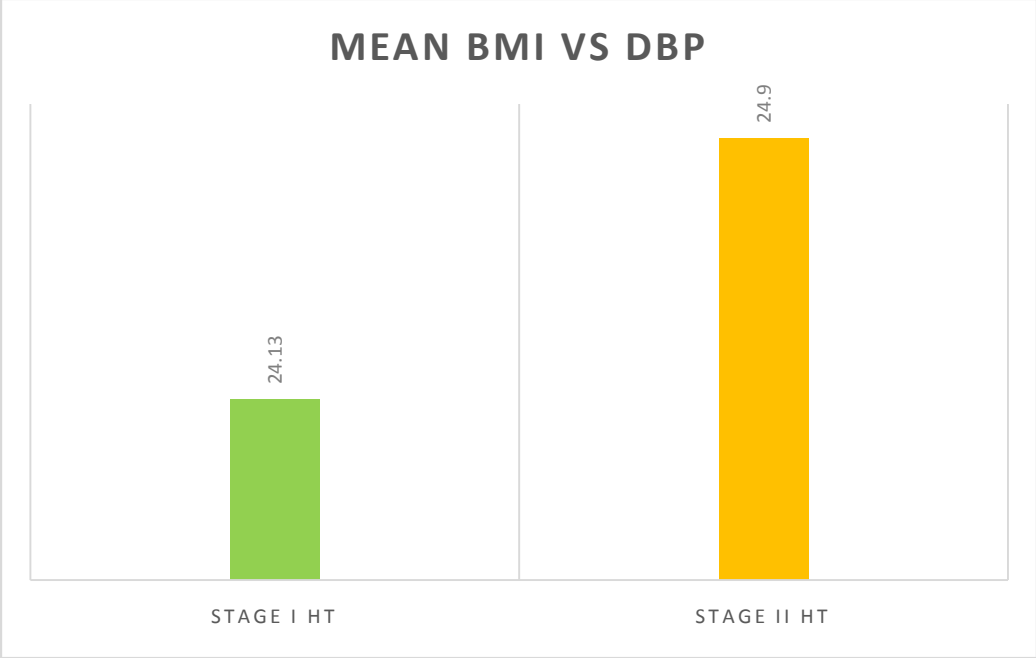


TABLE 12- COMPRISON OF MEAN SBP WITH BMI

BMI	SYSTOLIC BP	
	MEAN	SD
MORE THAN 25	150.1	19.23
LESS THAN 25	146.7	18.7
UNPAIRED T TEST		
P VALUE - 0.206		
NON SIGNIFICANT		

CHART 12- COMPARISON OF MEAN SBP WITH BMI

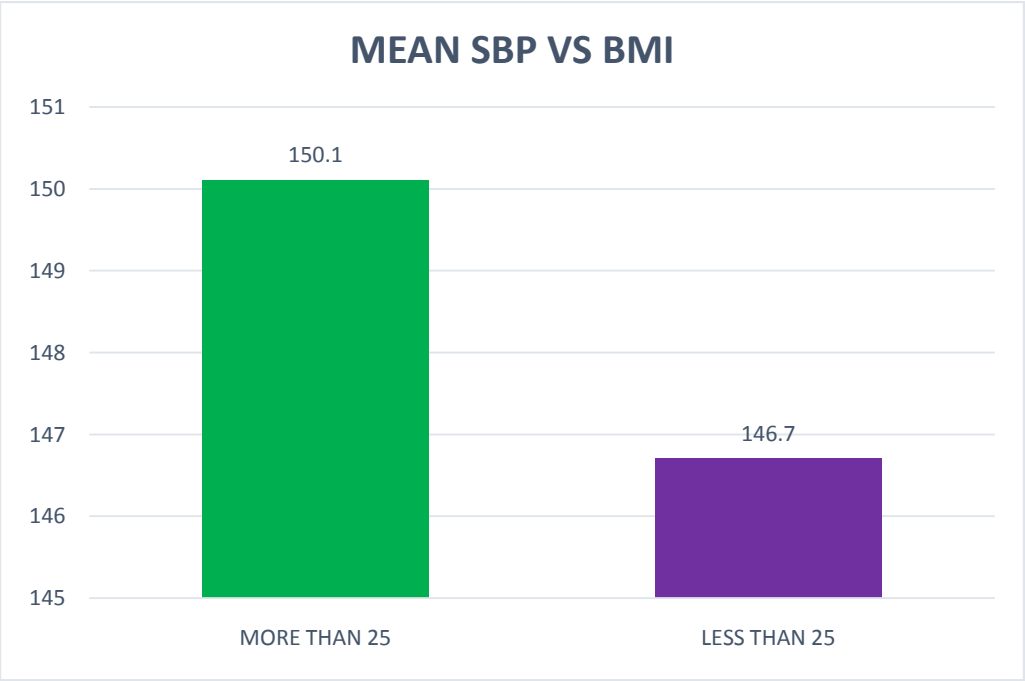


TABLE 13-COMPARISON OF MEAN DBP WITH BMI

BMI	DIASTOLIC BP	
	MEAN	SD
MORE THAN 25	99.48	11.84
LESS THAN 25	97.67	11.81
UNPAIRED T TEST		
P VALUE - 0.280		
NON SIGNIFICANT		

CHART 13-COMPARISON OF MEAN DBP WITH BMI

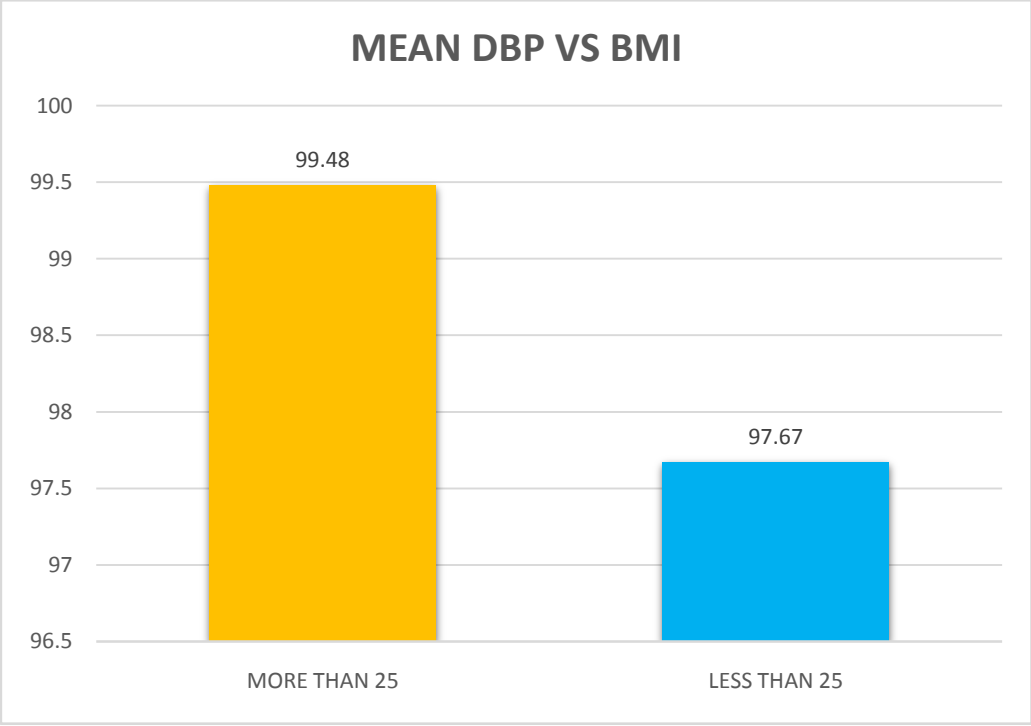


TABLE 14- HYPERTENSIVE RETINOPATHY DISTRIBUTION

FUNDAL CHANGES	NO OF PATIENTS	PERCENTAGE
PRESENT	41	20%
ABSENT	159	80%

CHART 14- HYPERTENSIVE RETINOPATHY DISTRIBUTION

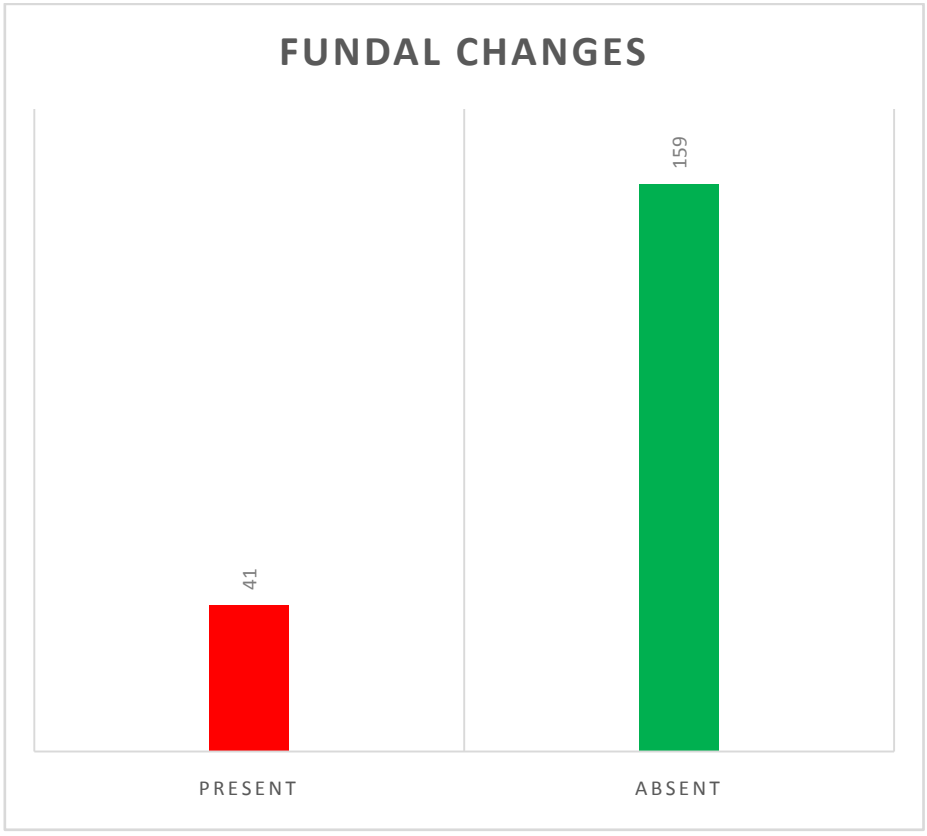


TABLE 15- GRADES OF RETINOPATHY

FUNDAL CHANGES	NO OF PATIENTS	PERCENTAGE
GRADE 1 HR	26	7%
GRADE 2 HR	15	13%
NORMAL	159	80%

CHART 15- GRADES OF RETINPATHY

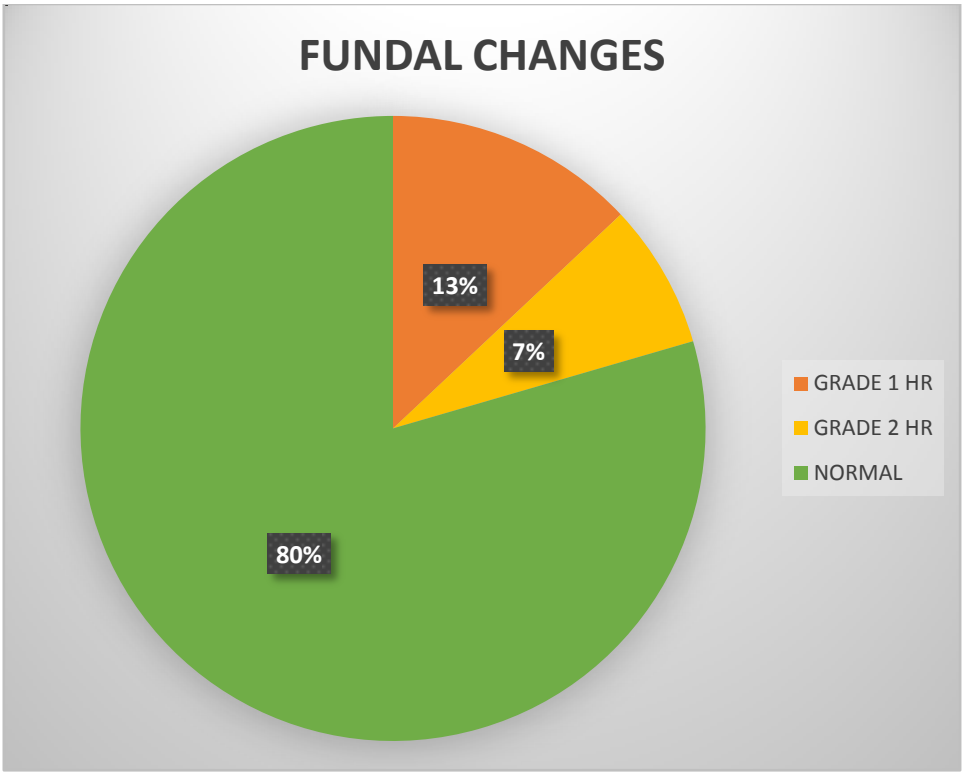


TABLE 16- COMPARISON OF STAGE OF SBP WITH HTN RETINOPATHY

FUNDUS - HR	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
PRESENT	0	41
ABSNET	53	106
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 16- COMPARISON OF STAGE OF SBP WITH HTN RETINOPATHY

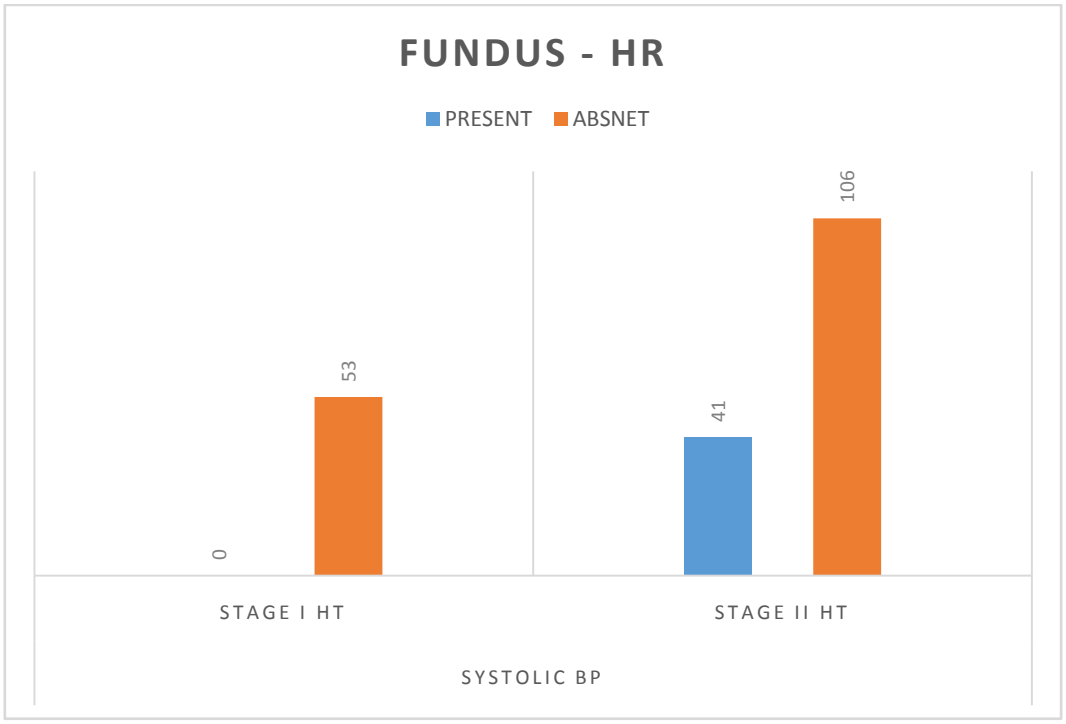


TABLE 17- COMPARISON OF STAGE OF SBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

FUNDUS	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
GRADE I HR	0	26
GRADE II HR	0	15
NORMAL	53	106
KRUSKAL WALLIS TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 17- COMPARISON OF STAGE OF SBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

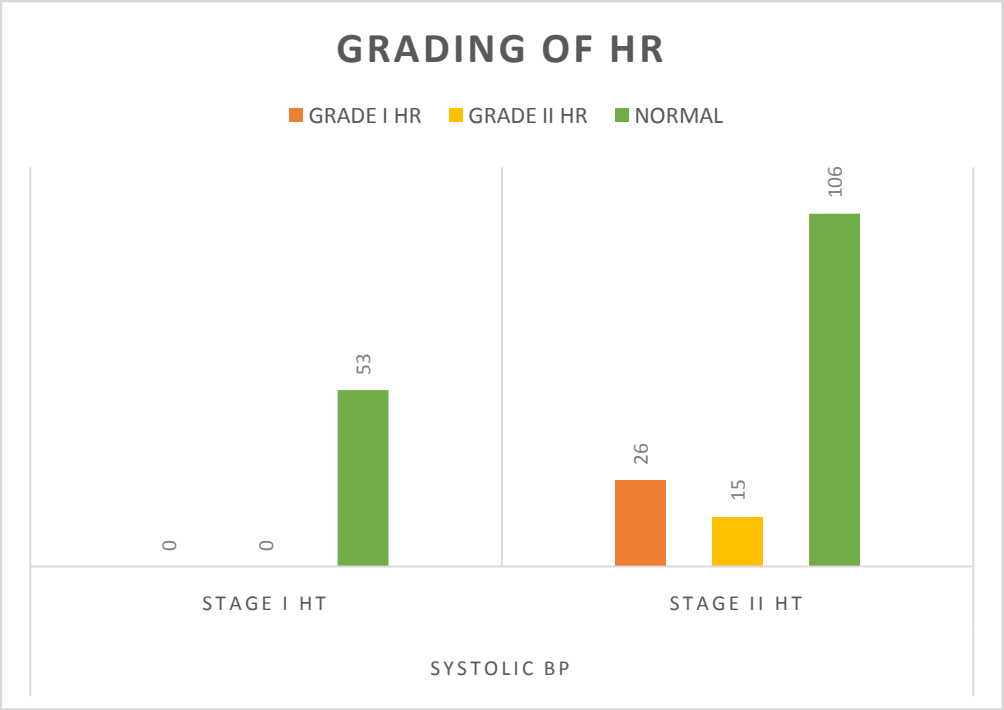


TABLE 18- COMPARISON OF STAGE OF DBP WITH HTN RETINOPATHY

FUNDUS - HR	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
PRESENT	0	41
ABSENT	8	151
CHI SQUARE TEST		
P VALUE - 0.143		
NON SIGNIFICANT		

CHART 18 - COMPARISON OF STAGE OF DBP WITH HTN RETINOPATHY

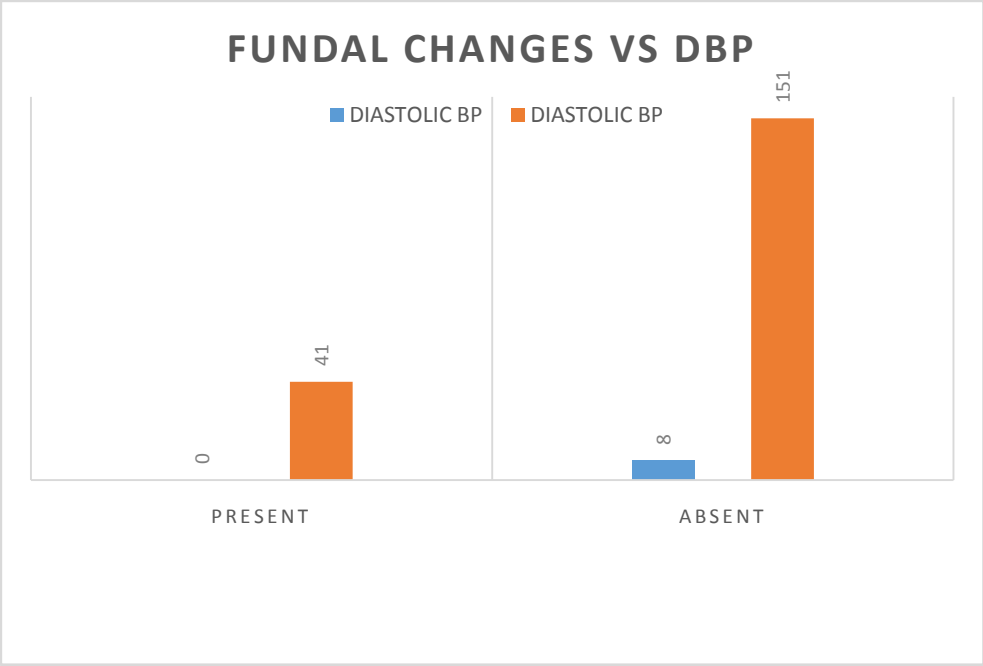


TABLE 19- COMPARISON OF STAGE OF DBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

FUNDUS	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
GRADE I HR	0	26
GRADE II HR	0	15
NORMAL	8	151
KRUSKAL WALLIS TEST		
P VALUE - 0.341		
NON SIGNIFICANT		

CHART 19- COMPARISON OF STAGE OF DBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

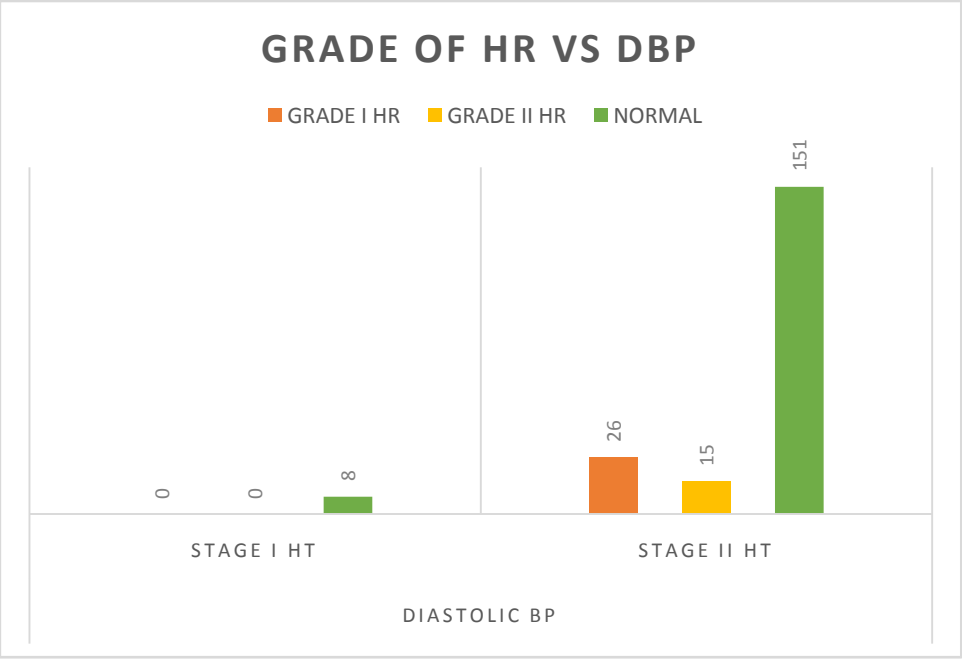


TABLE 20- COMPARISON OF MEAN SBP WITH HTN RETINOPATHY

FUNDUS - HR	SYSTOLIC BP	
	MEAN	SD
PRESENT	165.61	20.13
ABSENT	143.9	15.94
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 20- COMPARISON OF MEAN SBP WITH HTN RETINOPATHY

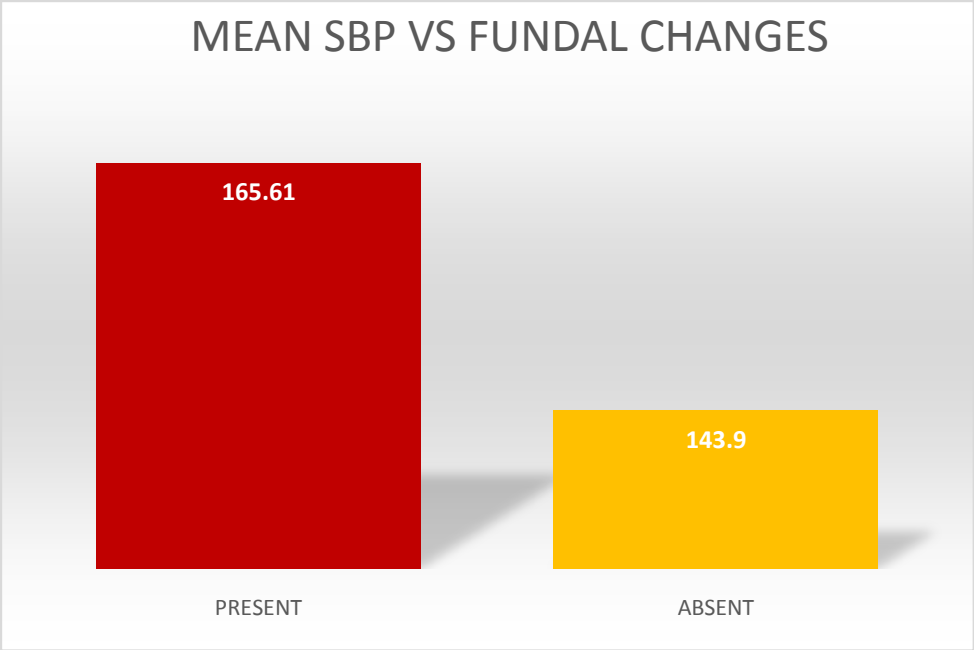


TABLE 21- COMPARISON OF MEAN DBP WITH HTN RETINOPATHY

FUNDUS - HR	DIASTOLIC BP	
	MEAN	SD
PRESENT	110.49	11.16
ABSENT	95.47	9.91
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 21- COMPARISON OF MEAN DBP WITH HTN RETINOPATHY

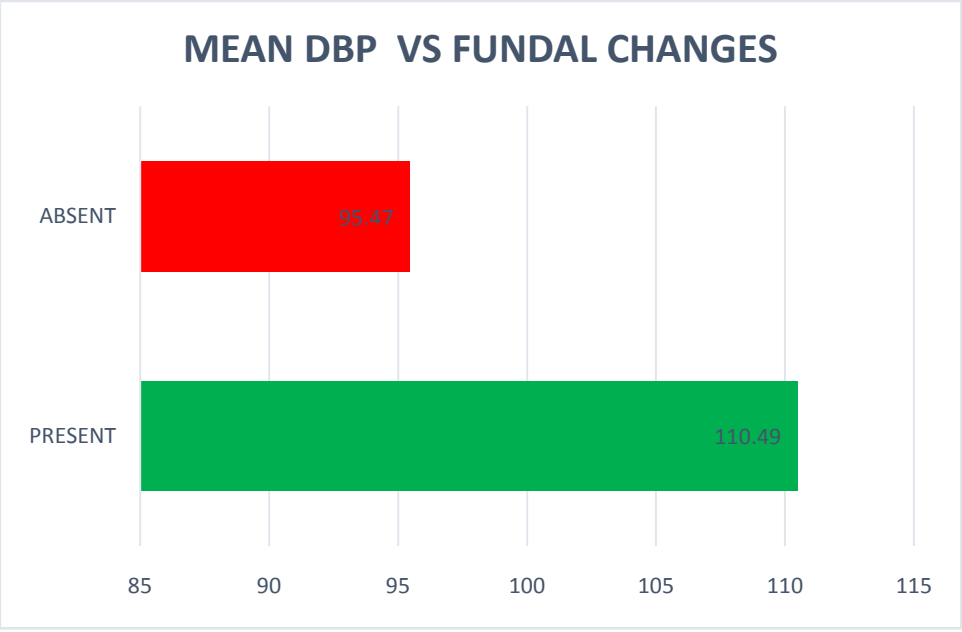


TABLE 22- COMPARISON OF MEAN SBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

FUNDUS GRADE	SYSTOLIC BP	
	MEAN	SD
GRADE I HR	156.92	13.49
GRADE II HR	180.67	21.2
NORMAL	143.9	15.94
ANOVA		
P VALUE - 0.001		
SIGNIFICANT		

CHART 22- COMPARISON OF MEAN SBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

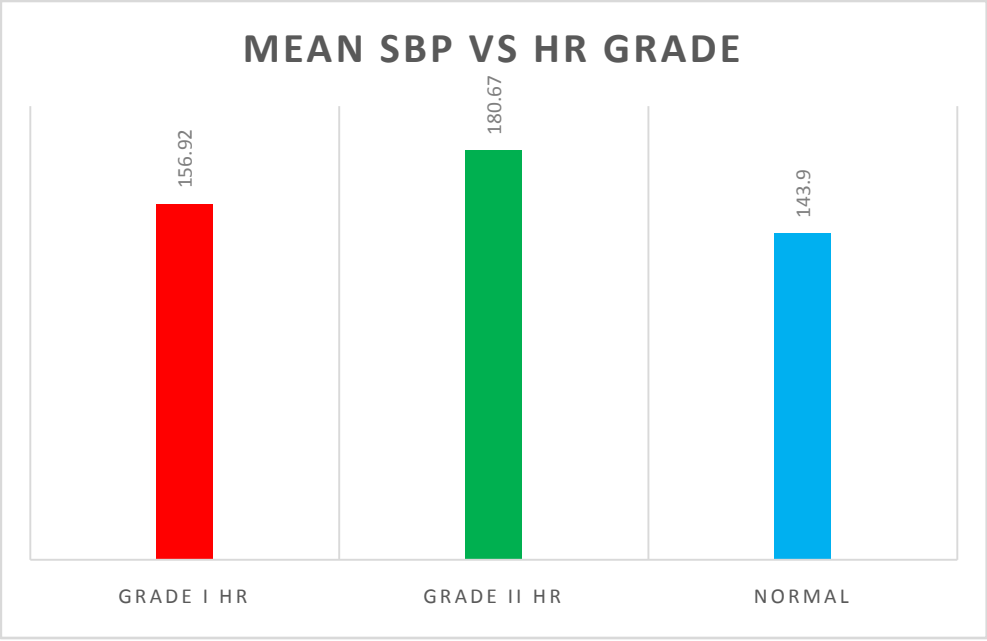


TABLE 23- COMPARISON OF MEAN DBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

FUNDUS GRADE	DIASTOLIC BP	
	MEAN	SD
GRADE I HR	106.54	10.17
GRADE II HR	117.33	9.61
NORMAL	95.47	9.91
ANOVA		
P VALUE - 0.001		
SIGNIFICANT		

CHART 23- COMPARISON OF MEAN DBP WITH GRADE OF HYPERTENSIVE RETINOPATHY

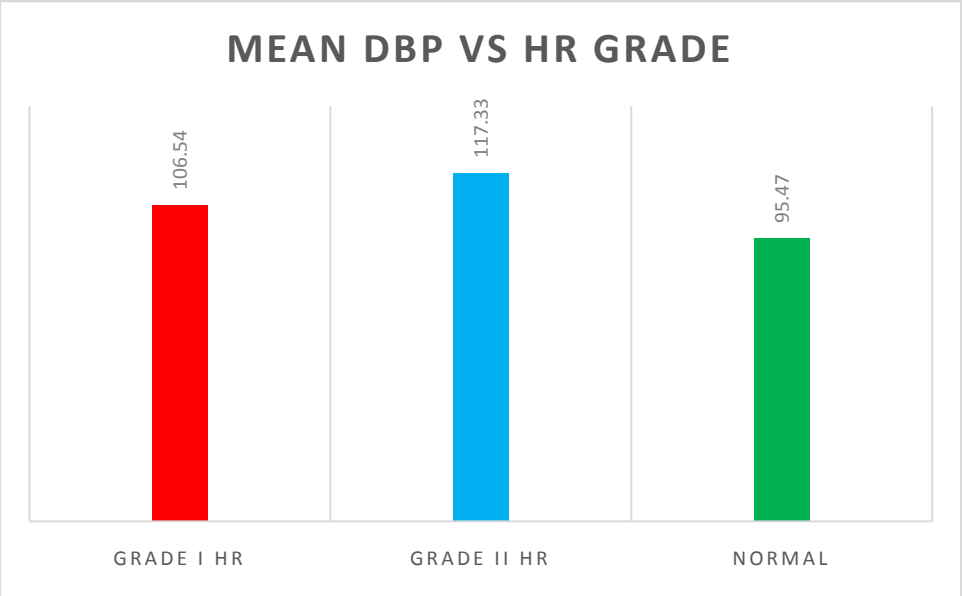


TABLE 24- DISTRIBUTION OF ALBUMINURIA

ALBUMINURIA	NO OF PATIENTS	PERCENTAGE
PRESENT	23	11%
ABSENT	177	89%

CHART 24- DISTRIBUTION OF ALBUMINURIA

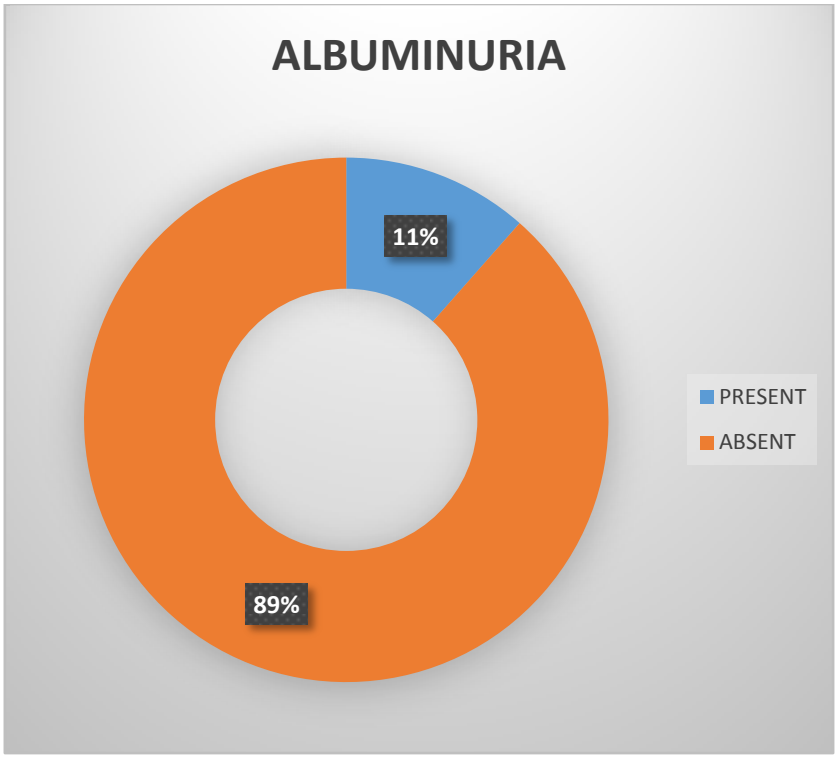


TABLE 25- GRADING OF ALBUMINURIA

ALBUMINURIA	NO OF PATIENTS	PERCENTAGE
1+	21	91%
2+	2	9%

CHART 25- GRADING OF ALBUMINURIA

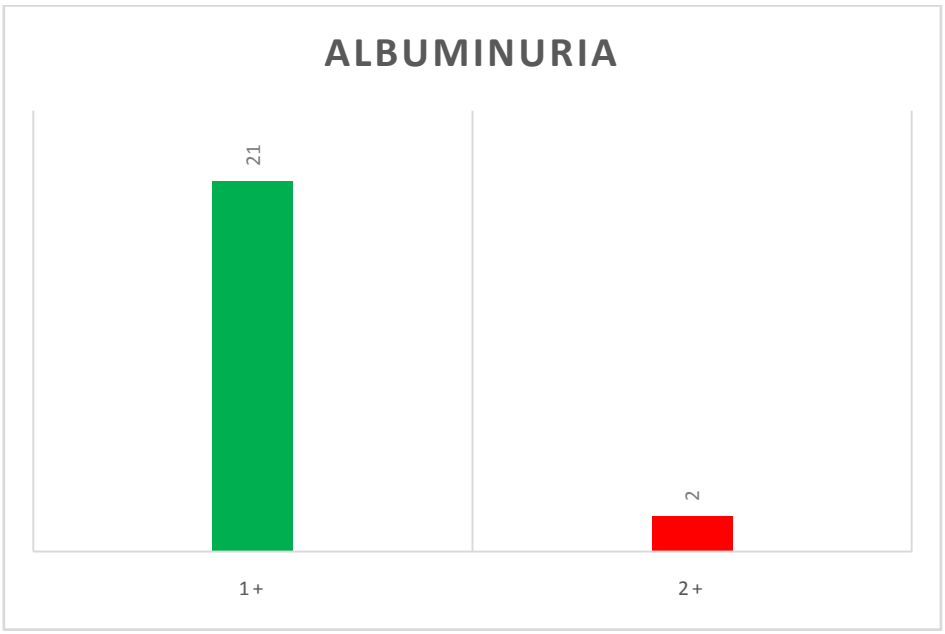


TABLE 26- COMPARISON OF STAGE OF SBP WITH ALBUMINURIA

ALBUMINURIA	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
PRESENT	2	21
ABSENT	51	126
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 26- COMPARISON OF STAGE OF SBP WITH ALBUMINURIA

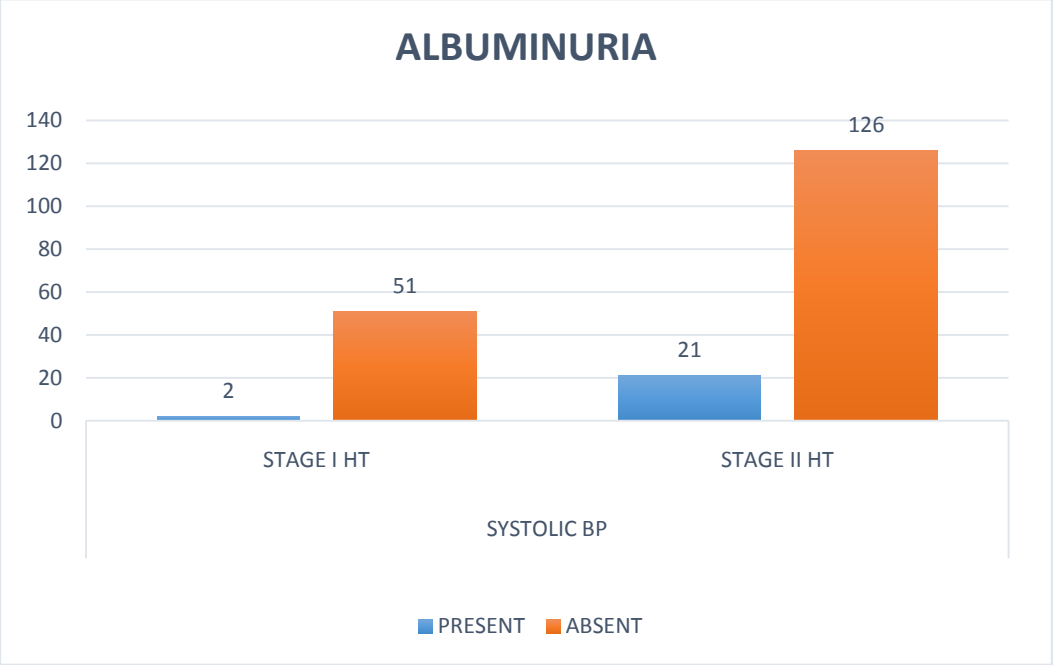


TABLE 27- COMPARISON OF STAGE OF DBP WITH ALBUMINURIA

ALBUMINURIA	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
PRESENT	1	22
ABSENT	7	170
CHI SQUARE TEST		
P VALUE - 0.928		
NON SIGNIFICANT		

CHART 27- COMPARISON OF STAGE OF DBP WITH ALBUMINURIA

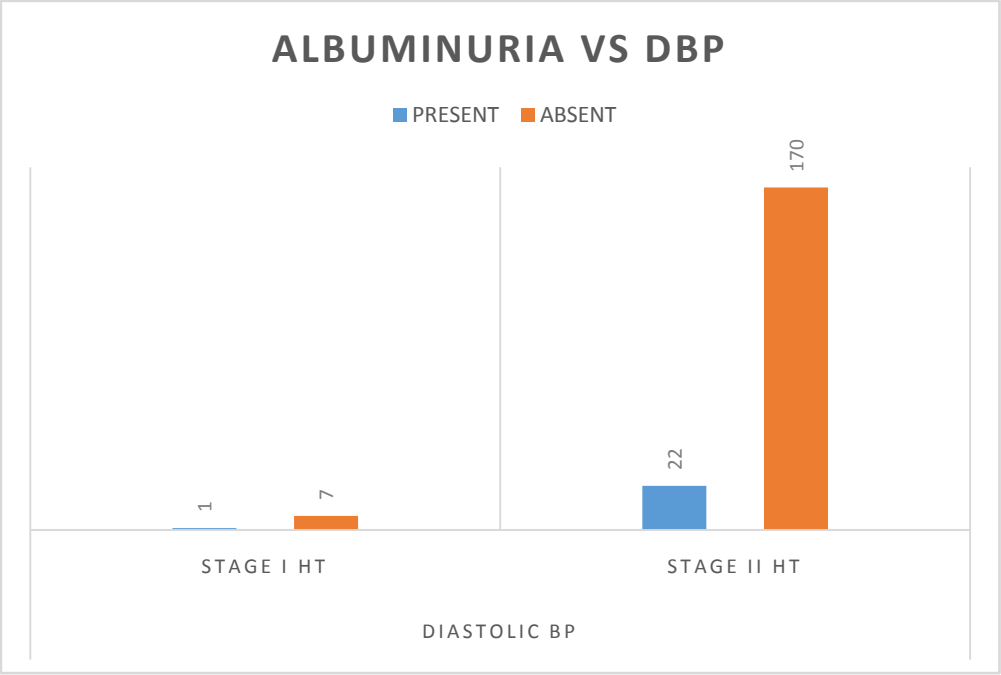


TABLE 28- COMPARISON OF MEAN SBP WITH ALBUMINURIA

ALBUMINURIA	SYSTOLIC BP	
	MEAN	SD
PRESENT	158.7	20.73
ABSENT	147.01	18.38
UNPAIRED T TEST		
P VALUE - 0.005		
SIGNIFICANT		

CHART 28 –COMPARISON OF MEAN SBP WITH ALBUMINURIA

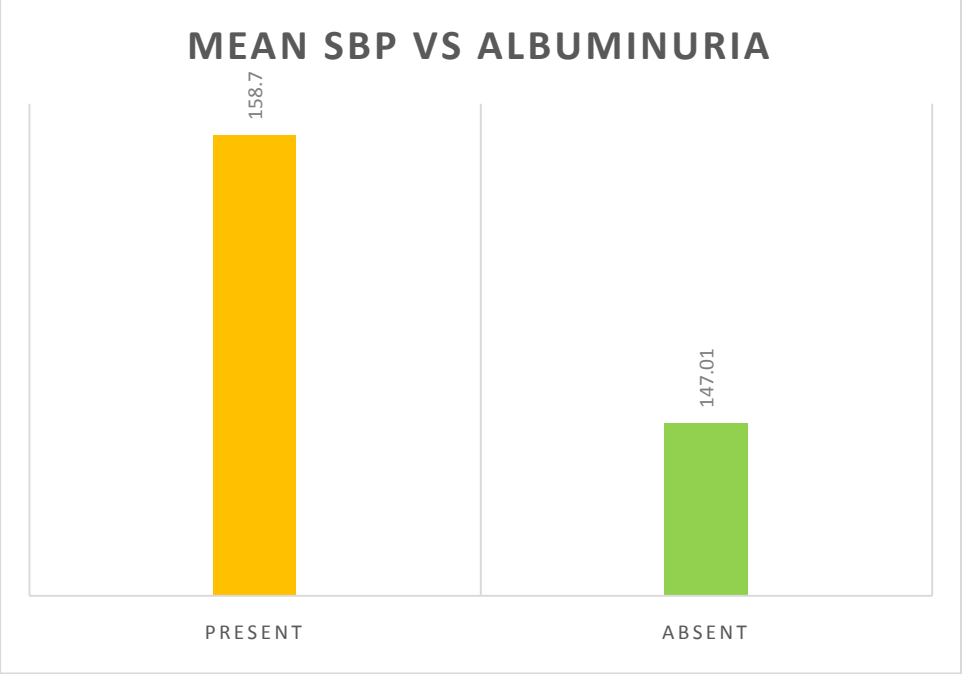


TABLE 29- COMPARISON OF MEAN DBP WITH ALBUMINURIA

ALBUMINURIA	DIASTOLIC BP	
	MEAN	SD
PRESENT	105.22	14.1
ABSENT	97.68	11.26
UNPAIRED T TEST		
P VALUE - 0.004		
SIGNIFICANT		

CHART 29 – COMPARISON OF MEAN DBP WITH ALBUMINURIA

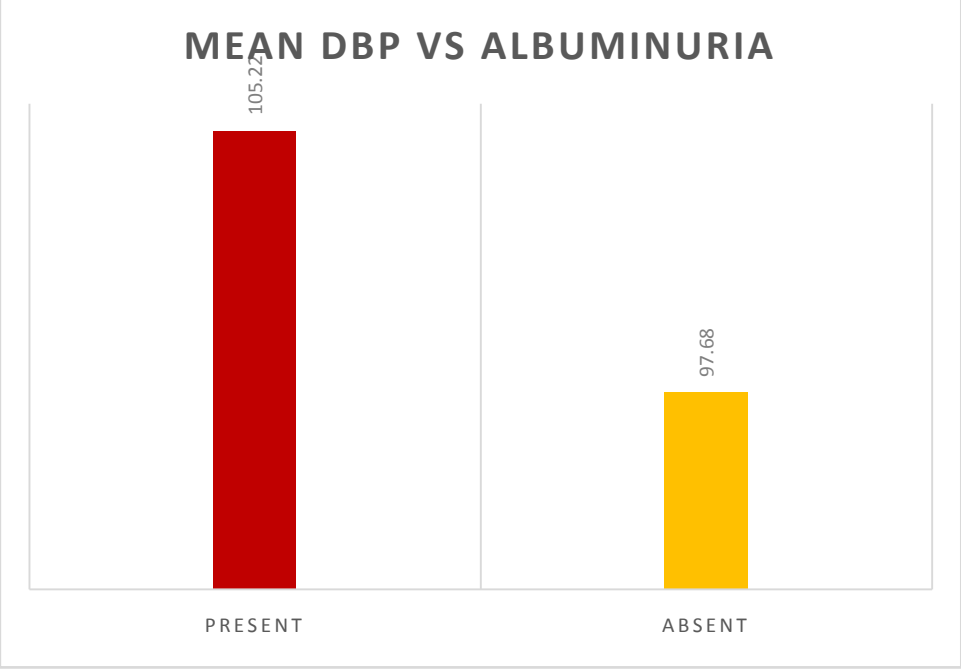


TABLE 30-SERUM CREATININE DISTRIBUTION

SERUM CREATININE	NO OF PATIENTS	PERCENTAGE
MORE THAN 1	52	26%
LESS THAN 1	148	74%

CHART 30- SERUM CREATININE DISTRIBUTION

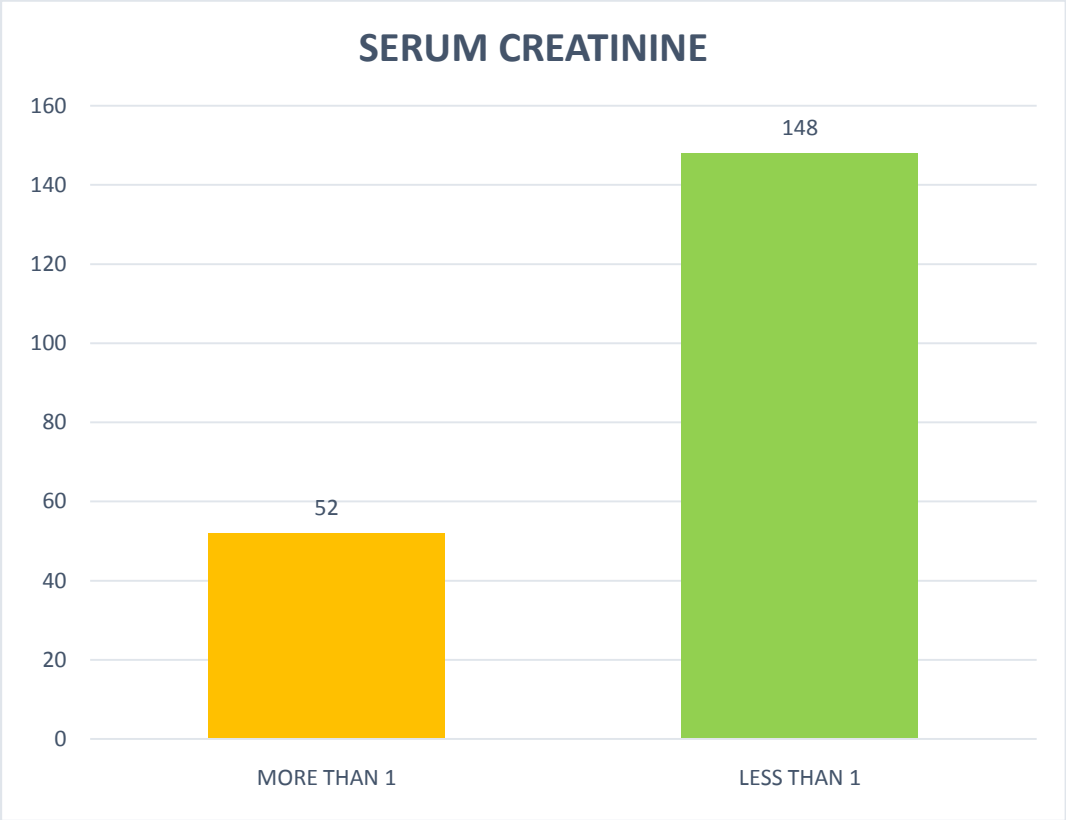


TABLE 31- COMPARISON BETWEEN STAGE OF SBP AND CREATININE

CREATININE	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
MORE THAN 1	12	40
LESS THAN 1	41	107
CHI SQUARE TEST		
P VALUE - 0.516		
NON SIGNIFICANT		

CHART 31- COMPARISON BETWEEN STAGE OF SBP AND CREATININE

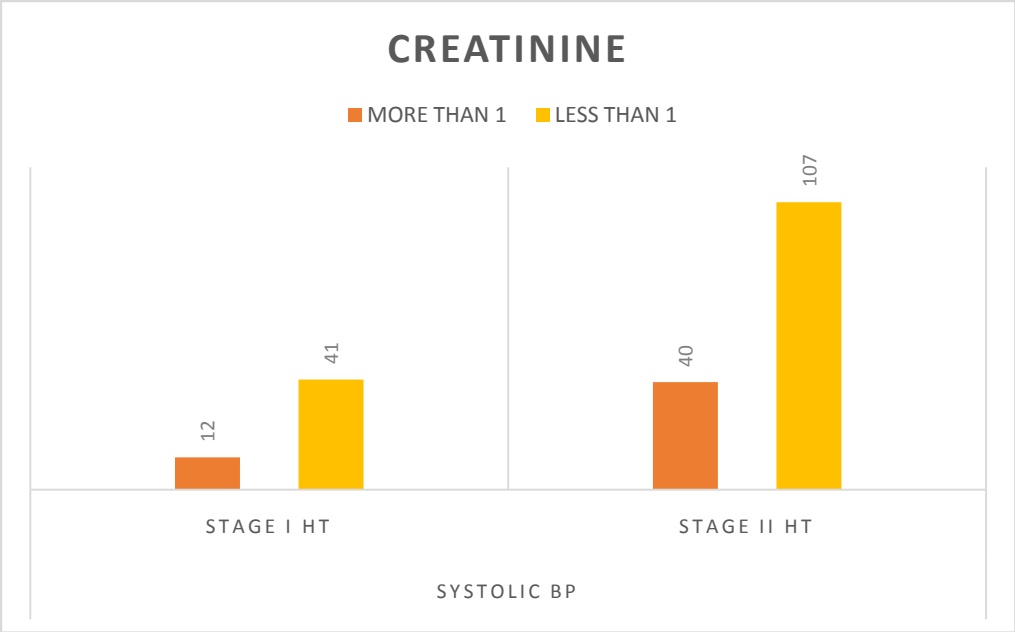


TABLE 32- COMPARISON BETWEEN STAGE OF DBP AND CREATININE

CREATININE	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
MORE THAN 1	2	50
LESS THAN 1	6	142
CHI SQUARE TEST		
P VALUE – 948		
NON SIGNIFICANT		

CHART 32- COMPARISON BETWEEN STAGE OF DBP AND CREATININE

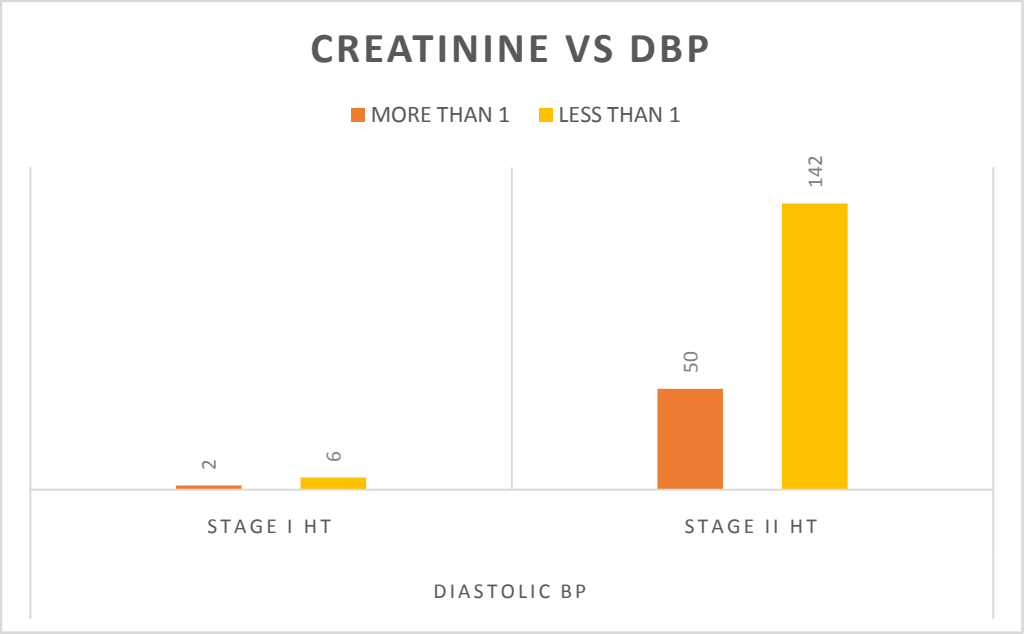


TABLE 33- COMPARISON OF MEAN SBP WITH SERUM CREATININE

CREATININE	SYSTOLIC BP	
	MEAN	SD
MORE THAN 1	148.27	17.68
LESS THAN 1	148.38	19.48
UNPAIRED T TEST		
P VALUE - 0.922		
NON SIGNIFICANT		

CHART 33- COMPARISON OF MEAN SBP WITH SERUM CREATININE

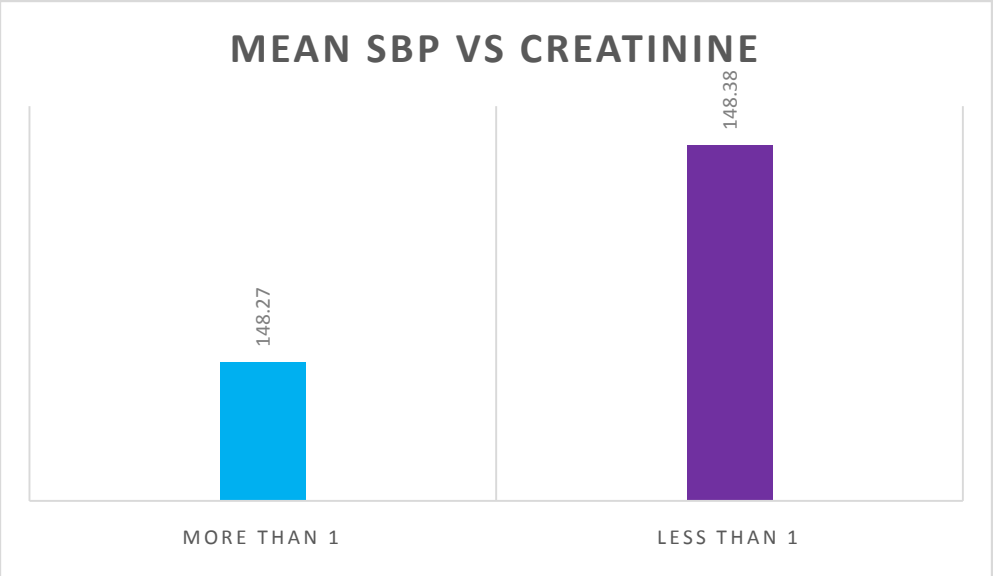


TABLE 34- COMPARISON OF MEAN DBP WITH SERUM CREATININE

CREATININE	DIASTOLIC BP	
	MEAN	SD
MORE THAN 1	99.23	12.65
LESS THAN 1	98.31	11.57
UNPAIRED T TEST		
P VALUE - 0.631		
NON SIGNIFICANT		

CHART 34- COMPARISON OF MEAN DBP WITH SERUM CREATININE

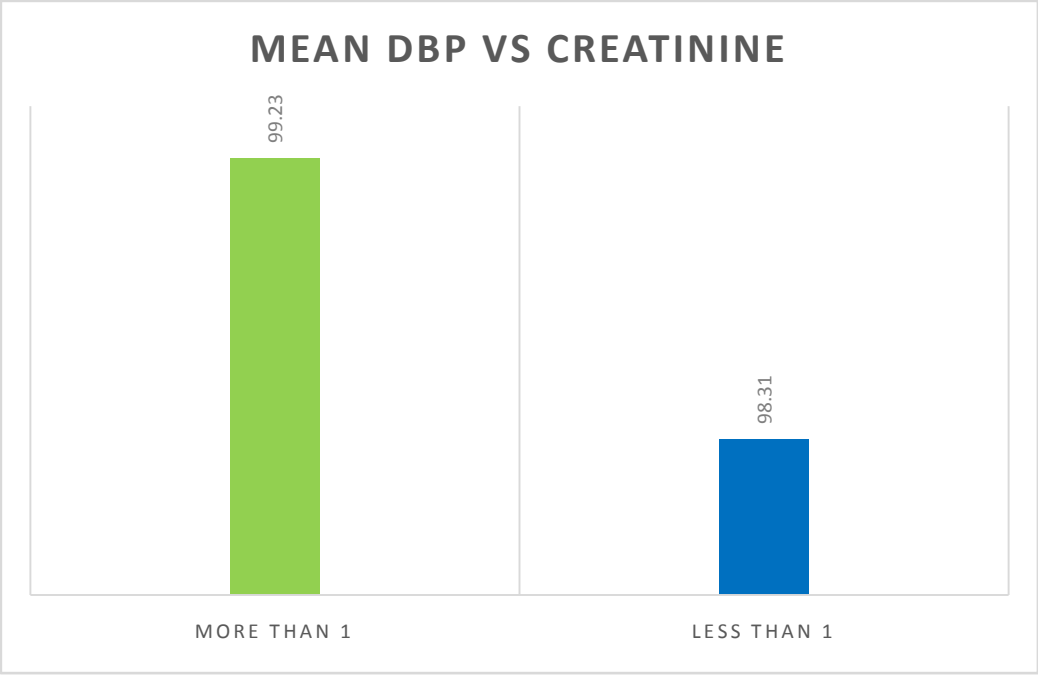


TABLE 35- ECG DISTRIBUTION

ECG	NO OF PATIENTS	PERCENTAGE
LVH	53	27%
NORMAL	147	73%

CHART 35 – ECG DISTRIBUTION

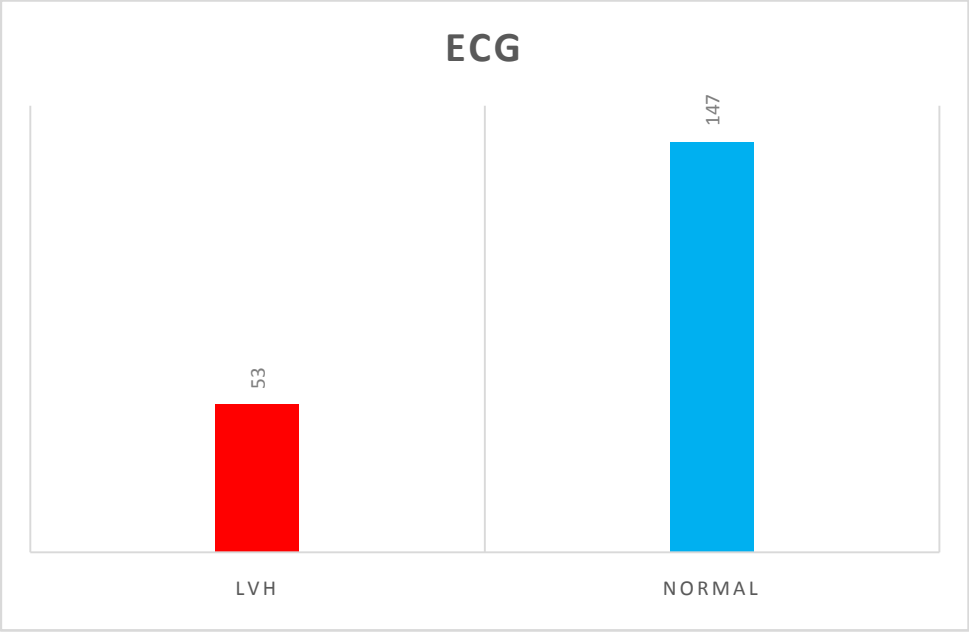


TABLE 36- COMPARISON OF STAGE OF SBP WITH ECG

ECG	SYSTOLIC BP	
	STAGE I HT	STAGE II HT
LVH	2	51
NORMAL	51	96
CHI SQUARE TEST		
P VALUE		
SIGNIFICANT		

CHART 36- COMPARISON OF STAGE OF SBP WITH ECG

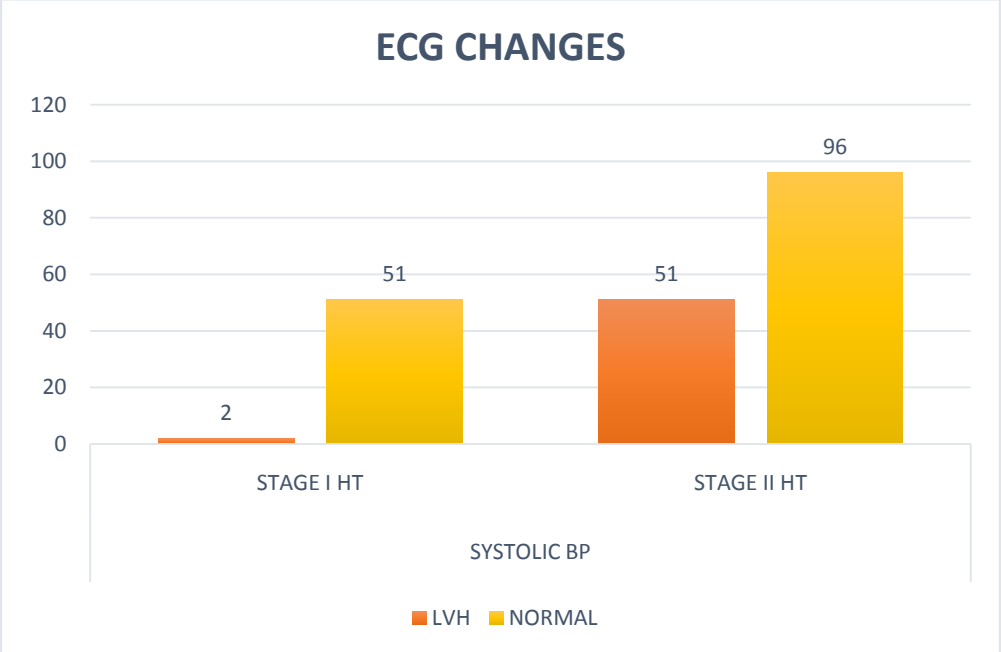


TABLE 37- TABLE 36- COMPARISON OF STAGE OF DBP WITH ECG

ECG	DIASTOLIC BP	
	STAGE I HT	STAGE II HT
LVH	0	53
NORMAL	8	139
CHI SQUARE TEST		
P VALUE		
SIGNIFICANT		

CHART 37 -TABLE 36- COMPARISON OF STAGE OF DBP WITH ECG

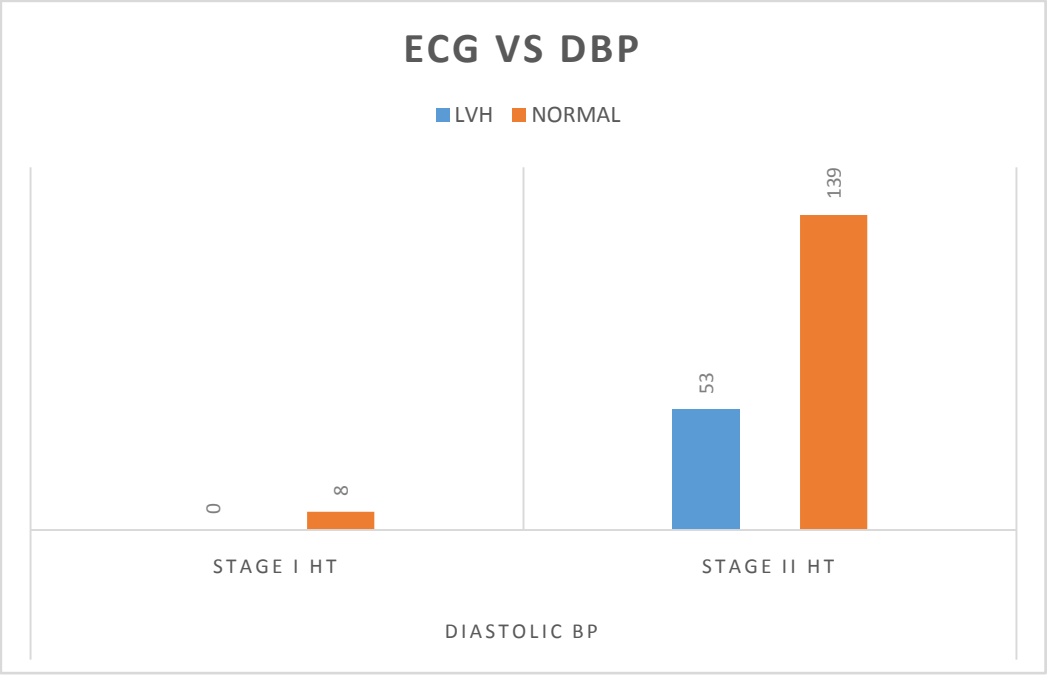


TABLE 38- COMPARISON OF MEAN SBP WITH ECG

ECG	SYSTOLIC BP	
	MEAN	SD
LVH	165.56	22.23
NORMAL	142.11	12.94
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 38- COMPARISON OF MEAN SBP WITH ECG

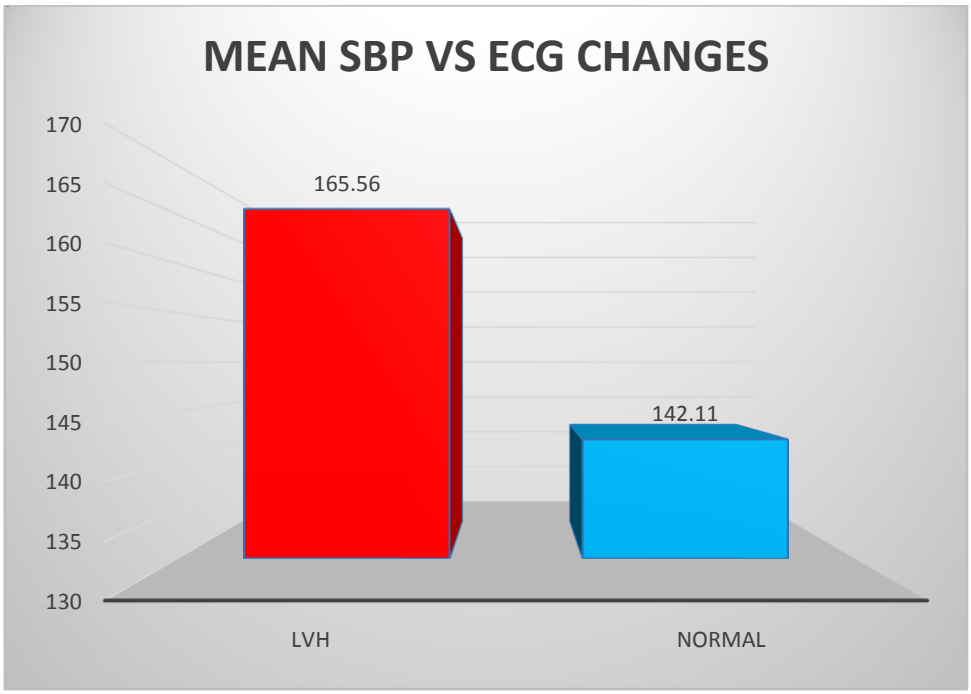


TABLE 39- COMPARISON OF DBP WITH ECG

ECG	DIASTOLIC BP	
	MEAN	SD
LVH	109.06	11.37
NORMAL	94.76	9.53
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART 39- COMPARISON OF MEAN DBP WITH ECG

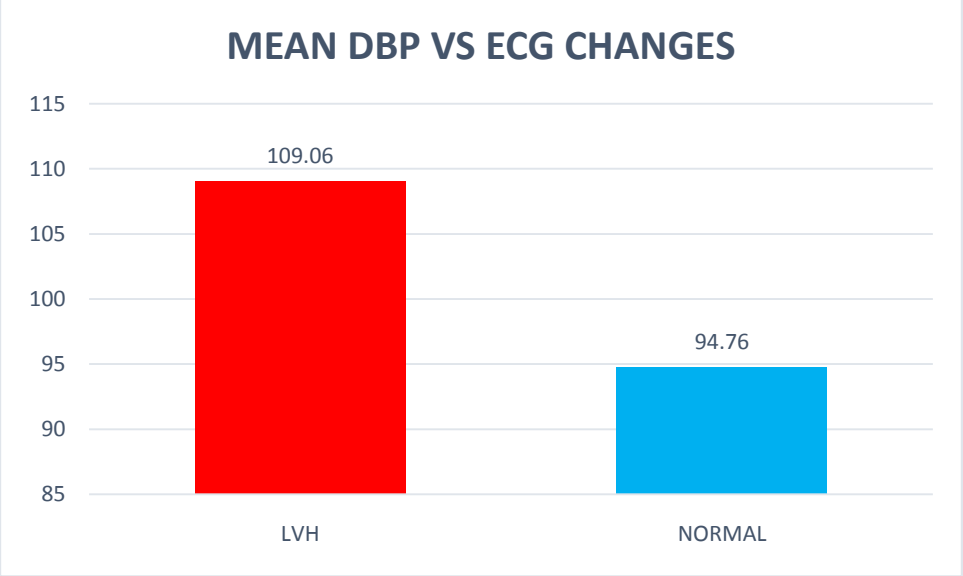


TABLE 40- FASTING BLOOD SUGAR DISTRIBUTION

FASTING BLOOD SUGAR	NO OF PATIENTS	PERCENTAGE
< 126	49	24%
>126	151	76%

CHART 40 – FASTING BLOOD SUGAR DISTRIBUTION

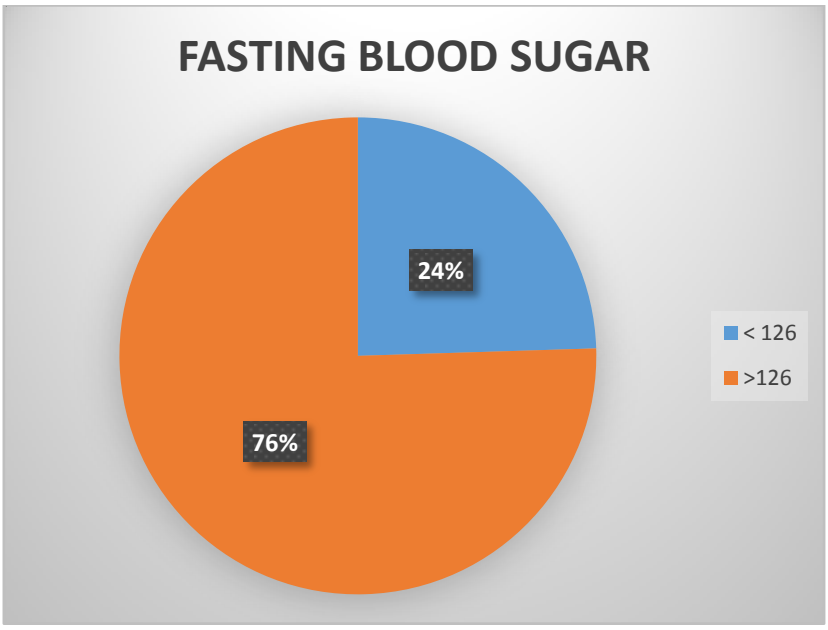


TABLE 41- NUMBER OF END ORGAN DAMAGE

NO. OF ORGAN DAMAGE	NUMBER OF PATIENTS	PERCENTAGE
0	121	60.5%
1	49	24.5%
2	22	11%
3	8	4%

TABLE 42- COMPARISON BETWEEN EYE CHANGES AND URINE ALBUMIN

FUNDUS	URINE ALBUMIN	NO. OF PATIENTS	PERCENTAGE
NORMAL	1+	6	3%
GRADE 1 HR	1+	4	2%
GRADE 2 HR	1+	13	6.5%

TABLE 43- COMPARISON BETWEEN EYE CHANGES AND ECG

FUNDUS	LVH	NUMBER OF PATIENTS	PERCENTAGE
GRADE 1 HR	PRESENT	12	6%
GRADE 2 HR	PRESENT	10	5%
GRADE 1 HR	ABSENT	14	7%
GRADE 2 HR	ABSENT	0	-

DISCUSSION

In this study, 85% of patients were more than 40 years and remaining 15% were less than 40 years. 23% were more than 60 years. Of which the elderly had more elevated systolic BP than younger age group. This is concurrent with other studies, PrakashkumarKyada et al has showed that isolated systolic hypertension is common in elderly (>60 years).

Out of the study population, 74% were having SBP in stage 2 with BP ≥140 mmHg at the time of diagnosis. 96% had DBP in stage 2 with BP ≥90 mmHg. The study included 48% males and 52% females. There is no significant difference in BP between both sexes based on stage of hypertension. Hayon Michelle Choi et al showed that in elderly age group females are more likely to be hypertensive patients.

When considering the BMI of these patients 48% had BMI more than 25 and there was no correlation between stage of hypertension and BMI in both systolic and diastolic blood pressure.

Assessment of end organ damage include hypertensive retinopathy in the form fundus examination, ECG to look for LVH based on Sokolov-Lyon index and urine albumin and serum creatinine to look for renal dysfunction.

20% had evidence of hypertensive retinopathy out of which 13% had grade 2 hypertensive retinopathy. Grade3 and grade 4 retinopathy were not seen any patient. RE Schmieder et al showed that 1% patients having hypertension have malignant hypertension and it has 3 year survival rate of 6%.

There was statistically significant correlation between stage of SBP and presence of hypertensive retinopathy with p value. This indicates higher the SBP more chance of hypertensive retinopathy based on Chi square test and KruskalWalhi test).

Grosso et al concluded that cardiovascular evaluation should be done in the presence of micro vascular changes in the retina. There was also statistically significant correlation between mean SBP and hypertensive retinopathy and as DBP is higher more chance of retinopathy. As the stage of hypertension progresses, the hypertensive retinopathy also progresses. This was statistically significant based on ANOVA test. Our study shows that there is correlation between BP recorded at the time of diagnosis and presence of hypertensive retinopathy for both systolic and diastolic blood pressure.

Roland E Schmieder showed that as stage of hypertension increases there is very significantly elevated risk of clinically manifest cardiovascular and renal disease. Gulhane Sanjay et al showed that 3% had retinopathy. Addo et al showed thata mean SBP and DBP were high if there is any organ damage compared to those without damage. 11% of newly detected hypertensives had albuminuria and 91% had 1+ proteinuria and 9% had 2+ proteinuria. Patients with stage 2 hypertension had more prevalence of albuminuria and it was statistically significant. Also the amount of albuminuria depends on mean systolic blood pressure. As mean SBP is more, albuminuria is more. It was statistically significant with p value of 0.005. Amount of albuminuria also corresponds to DBP. As DBP is more, risk of albuminuria. Yao Ping Lin

showed that albuminuria and eGFR<60 ml/min is associated with increased all cause and cardiovascular mortality.

26% of the patients have serum creatinine more than 1.0mg%. But the stage of hypertension had no correlation with serum creatinine levels. Also no significant correlation with serum creatinine levels. Also no significant correlation between mean SBP and DBP and serum creatinine. 27% had ECG changes of LVH according to Sokolov Lyon index. There was significant correlation between ECG and stage of hypertension with SBP, DBP and mean SBP and DBP. Systolic BP is an independent strong predictor of risk of cardiovascular and renal disease. He J et al showed that isolated systolic BP is the commonest type of hypertension in geriatric age group. LVH is the most common complication of hypertension in these patients.

79 patients had at least one end organ damage which is 24.5% and 121 patients did not have any organ damage which is 60.5%. There is 11% patients with evidence of two end organ damage out of three screened for. And 8% had all three end organ damage with retinopathy, nephropathy and hypertensive heart disease.

SUMMARY

This study shows that there is significant correlation between the magnitude of blood pressure at the time of diagnosis and prevalence of end organ damage. Higher the blood pressure at the time of diagnosis, more is the risk of presence of end organ damage at that time. In this study there is correlation between blood pressure at time of diagnosis and presence of retinopathy and hypertensive heart disease. Such patients have increased risk of other complications of hypertension leading to increased risk of cardiovascular mortality.

CONCLUSION

Hypertension is a silent killer disease and is on a raising trend in the current era. It has become the most common cause of cardiovascular events contributing to morbidity and mortality. In this study, as the magnitude of blood pressure is higher at the time of diagnosis there chance of presence of end organ damage. This signifies the importance of evaluating for all the end organ damage at the time of diagnosis of the disease. This reduces risk of morbidity and mortality and prevention of complications.

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PROFORMA

Name :

Age :

Sex :

O.P. No :

Occupation and address :

Complaints :

Symptom	Yes/ No	Duration
Vision disturbances		
Headache, vomiting		
Giddiness		
Epistaxis		
Chest pain, breathlessness		
Reduced urine output		
Fatigue		
Insomnia		

Past history: (any significant comorbid illness):

General examination:

System examination:

Assessment:

SBP		URINE ALBUMIN	
DBP		SERUM CREATININE	
HEIGHT		ELECTROCARDIOGRAM	
WEIGHT			
BMI		FBS	
FUNDUS		S.CHOLESTEROL	

MASTER CHART:

NAME	AGE	SEX	SBP	DBP	BMI	FUNDUS	U/A	S.Cr	ECG	FBS	S.TC
KRISHNAMOORTHY	31	M	140	100	21	NORMAL	NIL	1	NORMAL	95	160
RAJESH	32	M	160	120	34	NORMAL	NIL	1	NORMAL	116	246
ANAND	33	M	130	90	29	NORMAL	NIL	1.1	NORMAL	108	162
KALIYAMMAL	53	F	140	90	26	NORMAL	NIL	1.1	NORMAL	158	234
JOTHIMANI	48	F	140	90	24	NORMAL	NIL	1.1	NORMAL	88	128
KARUPPAMMAL	60	F	140	90	28	NORMAL	NIL	0.8	NORMAL	236	209
JOTHI	37	F	150	110	24	GRADE 1 HR	1+	1.8	NORMAL	90	170
RAJENDRAN	60	M	130	100	19	NORMAL	NIL	1	NORMAL	66	153
CHAKRAPANI	55	M	140	90	23	NORMAL	NIL	1.4	NORMAL	89	189
SAMPATH KUMAR	52	M	160	100	27	NORMAL	NIL	1.3	NORMAL	80	153
SHERLEY	40	F	170	110	36	NORMAL	NIL	0.9	NORMAL	87	189
NASEEMA	44	F	160	110	35	GRADE 2 HR	1+	1	LVH	96	181
MANONMANI	58	F	150	100	37	GRADE 1 HR	NIL	1	NORMAL	84	152
SARASWATHY	50	F	140	100	23	NORMAL	1+	1	LVH	230	186
SARASU	46	F	180	110	28	GARDE 2 HR	NIL	1	NORMAL	170	139
NOORJAHAN	50	F	140	90	20	NORMAL	NIL	1	NORMAL	100	226
REGINA	40	F	130	90	32	NORMAL	NIL	0.9	NORMAL	92	128
MARGARET	50	F	130	90	28	NORMAL	NIL	0.9	NORMAL	93	197
SHARADHA	44	F	140	110	29	GRADE 1 HR	NIL	0.7	NORMAL	100	228
SATHYAMOORTHY	58	M	160	100	25	NORMAL	1+	0.8	NORMAL	85	164
MATHAVI	70	F	180	100	19	NORMAL	NIL	0.8	LVH	71	138
ARULMANI	55	F	150	90	22	NORMAL	NIL	0.8	NORMAL	109	106
RAKKAMMAL	50	F	140	100	25	NORMAL	NIL	0.8	NORMAL	98	193
RAVIKUMAR	32	M	150	90	25	NORMAL	NIL	0.9	NORMAL	92	117
AROKKIYADHAS	63	M	140	110	22	NORMAL	NIL	0.7	LVH	90	174
KRISHNAN	62	M	140	90	25	NORMAL	NIL	1	NORMAL	98	158
PONNAMMAL	47	F	140	100	19	NORMAL	NIL	0.7	NORMAL	150	77
NAGARAJ	63	M	130	90	26	NORMAL	NIL	1	NORMAL	86	126
SUNDARAJ	57	M	130	100	27	NORMAL	NIL	0.8	LVH	240	134
MANI	56	M	180	130	22	GRADE 2 HR	NIL	5.8	LVH	98	79
MURUGAN	64	M	180	140	22	NORMAL	2+	1	NORMAL	95	202
VIJAYALAKSHMI	68	F	140	100	25	NORMAL	NIL	0.7	NORMAL	92	104
MURUGAYEE	48	F	130	90	27	NORMAL	NIL	0.9	NORMAL	94	208
HAJARA	52	F	180	140	26	GRADE 1 HR	NIL	1.2	NORMAL	108	217
SHANTHAMANI	48	F	140	100	29	NORMAL	1+	1	LVH	120	159
THANGAVELU	60	M	150	110	26	GRADE 1 HR	NIL	1	NORMAL	102	149
SANTHAMANI	60	F	150	100	24	GRADE 1 HR	NIL	0.9	NORMAL	160	127
RAVI	50	M	130	90	19	NORMAL	NIL	0.9	NORMAL	92	187
LAKSHMI	62	F	160	100	22	NORMAL	NIL	0.9	LVH	240	126
APPAVU	68	M	140	90	29	NORMAL	NIL	1	NORMAL	98	117
SEENIYAPPAN	65	M	150	100	24	GRADE 1 HR	NIL	0.7	NORMAL	94	570
KUMAR	50	M	130	90	23	NORMAL	NIL	1	NORMAL	96	155
ANTONY	63	M	140	90	29	NORMAL	NIL	1	NORMAL	94	167
SELVARAJ	57	M	160	100	27	NORMAL	NIL	1.9	LVH	162	197
DILSATH	36	F	190	120	29	NORMAL	1+	0.9	LVH	80	164
SELVARAJ	45	M	200	120	24	GRADE 2 HR	NIL	0.9	NORMAL	81	148
VISHWANATHAN	46	M	160	120	27	NORMAL	NIL	1.1	NORMAL	130	175
VIJAYA	63	F	160	100	27	NORMAL	NIL	0.8	LVH	130	175
NATRAJ	55	M	140	100	29	NORMAL	NIL	0.9	NORMAL	71	179
RAJESHWARI	44	F	160	100	33	NORMAL	1+	0.7	NORMAL	92	160

KANAGA	56	F	130	90	26	NORMAL	NIL	0.8	NORMAL	90	92
BRINDHA DEVI	56	F	140	90	36	NORMAL	NIL	0.8	NORMAL	204	229
GOPALAKRISHNAN	38	M	130	90	24	NORMAL	NIL	4.8	NORMAL	180	231
KANNAN	39	M	130	100	25	NORMAL	NIL	0.9	NORMAL	260	221
PALANISAMY	67	M	130	90	25	NORMAL	NIL	1	NORMAL	89	276
JAYALAKSHMI	50	F	200	110	20	NORMAL	NIL	0.8	LVH	98	229
SAVITHRI	40		140	100	36	NORMAL	NIL	1.2	NORMAL	240	226
SARASWATHY	63	F	140	90	27	NORMAL	NIL	0.9	NORMAL	120	211
FATHIMABANU	50	F	140	90	23	NORMAL	NIL	0.8	NORMAL	98	219
KANNAMMAL	62	F	150	100	23	GRADE 1 HR	NIL	0.6	NORMAL	92	353
VALLIYAMMAL	64	F	140	90	24	NORMAL	NIL	0.8	NORMAL	73	204
RAO	36	M	130	100	24	NORMAL	NIL	0.9	NORMAL	88	190
MUTHUKUMAR	50	M	150	90	25	NORMAL	NIL	1.4	NORMAL	89	138
RATHINAVEL	49	M	130	90	25	NORMAL	NIL	0.8	NORMAL	99	204
RAJAN	64	M	140	100	24	GRADE 1 HR	NIL	0.8	LVH	95	276
SUBBAMMAL	56	F	160	120	30	NORMAL	NIL	1.2	LVH	85	174
KALAVATHY	60	F	150	90	26	NORMAL	NIL	0.7	NORMAL	99	186
IQBAL	52	M	150	90	25	NORMAL	NIL	0.9	NORMAL	100	179
FATHIMA	48	F	130	80	23	NORMAL	NIL	1	NORMAL	92	180
JUSTIN	52	M	220	120	27	GRADE 2 HR	NIL	0.8	LVH	100	219
IMAM	43	M	220	130	25	NORMAL	NIL	1	LVH	96	196
VELUMANI	48	F	200	130	24	GRADE 2 HR	1+	0.7	LVH	97	175
POUNRAJ	62	M	150	90	22	NORMAL	NIL	0.9	NORMAL	94	151
ASIYA	54	F	140	100	28	GRADE 1 HR	1+	1	NORMAL	240	147
VELLINGIRI	75	M	170	100	23	NORMAL	NIL	0.9	LVH	98	176
JAMILA	46	F	140	90	29	NORMAL	1+	0.9	LVH	260	206
KUMAR	37	M	150	100	19	NORMAL	NIL	1.1	NORMAL	77	192
GEETHA	45	F	150	100	27	NORMAL	NIL	2	LVH	98	238
JOHNSON	45	M	130	90	19	NORMAL	NIL	1.5	NORMAL	84	144
RANGASAMY	46	M	150	100	19	GRADE 1 HR	1+	1	NORMAL	210	210
KITTUSAMY	60	M	140	90	19	NORMAL	NIL	0.7	NORMAL	95	191
MURUGASAMY	65	M	160	110	19	NORMAL	NIL	1.2	LVH	370	177
RAJI	51	F	130	90	30	NORMAL	NIL	1	NORMAL	100	228
SUBBULAKSHMI	55	F	130	90	22	NORMAL	NIL	1	NORMAL	190	380
THIRUMOORTHY	53	M	150	90	24	NORMAL	NIL	0.8	NORMAL	204	197
SAKUNTHALA	65	F	140	100	27	NORMAL	NIL	1.1	NORMAL	98	212
ABDUL RAHMAN	48	M	140	90	24	NORMAL	NIL	1.8	NORMAL	96	158
KADHAR BEEVI	50	F	160	110	28	GRADE 1 HR	1+	1	LVH	100	188
SABIRA	49	F	140	110	26	NORMAL	NIL	1.2	LVH	112	164
USAIN	48	M	160	120	35	GRADE 2 HR	NIL	1	LVH	98	132
NAJUMUNISHA	40	F	130	90	24	NORMAL	NIL	1.1	NORMAL	95	385
AMUTHA	48	F	130	90	20	NORMAL	NIL	0.9	NORMAL	130	194
ANTONY	47	M	150	100	24	GRADE 2 HR	NIL	0.7	NORMAL	90	185
MAGESH	40	M	140	90	28	NORMAL	NIL	1	NORMAL	100	160
RAMASAMY	73	M	140	90	19	NORMAL	NIL	1.4	NORMAL	98	166
RAJAMANI	55	M	180	110	26	GRADE 1 HR	NIL	0.9	LVH	170	277
SELVARAJ	51	M	150	100	24	NORMAL	NIL	1	NORMAL	112	233
GNANADEVAN	40	M	130	90	22	NORMAL	NIL	1	NORMAL	230	213
KAMALAM	50	F	130	90	27	NORMAL	NIL	0.8	NORMAL	84	194
SARASWATHI	55	F	170	100	22	NORMAL	NIL	1	LVH	98	281
AMBIKA	50	F	160	110	28	NORMAL	1+	0.9	LVH	200	239
ANJALA	50	F	180	110	27	GRADE 2 HR	NIL	0.8	NORMAL	96	279
NAGARAJ	48	M	140	100	19	NORMAL	NIL	1	NORMAL	97	220

SUBRAMANI	50	M	130	80	29	NORMAL	NIL	1.1	NORMAL	380	191
MUTHU	64	M	160	120	21	GRADE 1 HR		0.9	LVH	92	195
CHANDRAKUMAR	43	M	150	90	28	NORMAL	1+	1.1	NORMAL	306	380
JAYANATH	38	F	140	90	27	NORMAL	NIL	0.7	NORMAL	86	141
PRABHUDASAN	50	M	160	110	32	GRADE 1 HR	NIL	1	LVH	88	184
SHANTHA	76	F	140	90	24	NORMAL	NIL	0.7	NORMAL	100	165
RAMAN	65	M	160	100	25	NORMAL	NIL	1.1	LVH	86	166
DEVAKI	55	F	160	100	21	NORMAL	NIL	0.7	NORMAL	88	172
RUCKMANI	40	F	180	130	26	GRADE 2 HR	NIL	0.7	LVH	92	209
DHARMARAJ	48	M	170	110	20	NORMAL	NIL	1.2	LVH	84	140
JAYAMANI	65	F	160	100	25	NORMAL	NIL	1	NORMAL	94	226
SURESH	32	M	130	90	27	NORMAL	NIL	1	NORMAL	91	216
RAMESH	52	M	140	100	23	NORMAL	NIL	1.7	NORMAL	89	182
SAMIAPPAN	52	M	140	90	19	NORMAL	NIL	1.4	NORMAL	260	186
JOTHI	58	F	140	90	24	NORMAL	NIL	0.6	NORMAL	98	184
NAGARAJ	61	M	130	100	23	NORMAL	NIL	0.9	NORMAL	99	198
KRISHNASAMY	72	M	150	90	23	NORMAL	NIL	1.7	NORMAL	92	190
KUMAR	51	M	130	100	22	NORMAL	NIL	0.9	NORMAL	96	189
JEYANTHI	42	F	130	90	19	NORMAL	NIL	1.1	NORMAL	374	239
AMUTHA	55	F	160	110	25	GRADE 1 HR	NIL	1.1	LVH	180	270
MURUGARAJ	53	M	160	110	37	NORMAL	NIL	1	NORMAL	98	179
BELSIYA	34	F	130	90	36	NORMAL	NIL	0.9	NORMAL	88	134
THANGAPANDI	62	M	160	120	22	GRADE 2 HR	NIL	0.9	NORMAL	104	182
ANTHONYSAMY	51	M	200	140	20	NORMAL	NIL	1	LVH	112	244
SUNDARAMMAL	40	M	160	120	22	GRADE 1 HR	NIL	2.9	LVH	97	239
SATHYAMOORTHY'	45	M	140	90	24	NORMAL	NIL	0.8	NORMAL	89	192
SALEEM	45	M	150	100	28	NORMAL	NIL	1	NORMAL	94	260
PUSPHALATHA	45	F	130	90	20	NORMAL	NIL	0.9	NORMAL	320	145
AYYAMMAL	70	F	140	80	19	NORMAL	NIL	0.8	NORMAL	81	182
MARUTHACHALAM	68	M	150	80	25	NORMAL	NIL	0.8	NORMAL	98	130
ILIYAS	64	M	130	100	19	NORMAL	NIL	0.9	NORMAL	92	198
APPASAMY	35	M	150	100	19	NORMAL	NIL	1	LVH	92	169
REGINA	33	F	160	80	27	NORMAL	NIL	0.9	NORMAL	75	214
RAJESHWARI	70	F	150	100	21	GRADE 1 HR	NIL	0.9	LVH	190	268
BAGYALAKSHMI	56	F	130	90	23	NORMAL	NIL	1.5	NORMAL	83	170
PUSPHA	67	F	170	120	22	GRADE 2 HR	1+	1	LVH	92	172
PARIMALA	50	F	130	90	22	NORMAL	NIL	0.9	NORMAL	100	256
THULASI	40	F	180	100	27	NORMAL	NIL	1	LVH	98	222
RABIYA	43	F	160	100	36	GRADE 1 HR	NIL	0.8	NORMAL	92	218
DHANALAKSHMI	55	F	150	90	30	NORMAL	NIL	0.9	NORMAL	94	139
FIROJA	45	F	130	90	25	NORMAL	NIL	0.8	NORMAL	280	213
SIRUBAI	48	F	140	90	20	NORMAL	NIL	1.1	NORMAL	90	160
TAMILARASI	46	F	150	90	25	GRADE 1 HR	1+	1	NORMAL	280	155
ARULRAJ	59	M	130	90	26	NORMAL	NIL	1.1	NORMAL	270	369
PARAMESHWARN	67	M	130	90	27	NORMAL	NIL	1	NORMAL	190	186
MEENATCHI	66	F	180	80	29	NORMAL	NIL	1	NORMAL	100	183
SIVAGAMI	50	F	130	100	24	NORMAL	NIL	1	LVH	92	121
JANAKI	45	F	130	90	20	NORMAL	NIL	0.7	NORMAL	95	211
RAMALINGAM	57	M	150	100	22	GRADE 1 HR	NIL	0.5	NORMAL	100	218
KAMARNISHA	40	F	130	90	37	NORMAL	NIL	1.4	NORMAL	84	88
SARAVANAN	45	M	130	90	19	NORMAL	NIL	1	NORMAL	93	179
BACKIYAMUTHU	53	M	200	120	19	GRADE 2 HR	NIL	1.1	LVH	73	318
KAMALAM	70	F	140	90	23	NORMAL	NIL	1.4	NORMAL	96	261

SAHAYAJACOB	47	M	130	80	20	NORMAL	1+	1.2	NORMAL	370	120
DHAMYANTHI	42	F	130	90	23	NORMAL	NIL	1.6	NORMAL	90	196
LEOPATRICK	48	M	130	90	28	NORMAL	NIL	0.9	NORMAL	88	142
ARUNACHALAM	70	M	130	90	19	NORMAL	NIL	1	NORMAL	250	180
SUMATHI	35	F	130	100	27	NORMAL	1+	0.9	NORMAL	340	190
MICHAELSAMY	63	M	140	90	23	NORMAL	NIL	1	NORMAL	94	131
MUTHAMMAL	65	F	130	90	21	NORMAL	NIL	0.8	NORMAL	92	130
GIRIJADEVI	45	F	140	90	19	NORMAL	NIL	0.6	NORMAL	80	298
NEBISHA	55	F	130	90	25	NORMAL	NIL	0.7	NORMAL	94	190
SEEMAN	65	M	140	90	19	NORMAL	NIL	1	LVH	76	255
GUNASUNDARI	42	F	150	100	25	NORMAL	NIL	0.9	NORMAL	79	243
RAJAMMAL	57	F	140	90	20	NORMAL	NIL	0.8	NORMAL	94	224
KRISHNAMOORTHY	46	M	130	90	20	NORMAL	NIL	1.4	NORMAL	92	145
MATHIANTONY	38	M	130	80	21	NORMAL	NIL	1	NORMAL	90	185
RAJESHKUMAR	31	M	140	110	34	NORMAL	NIL	1	NORMAL	116	186
VASANTHAMANI	43	F	150	100	36	GRADE 1 HR	NIL	1.4	NORMAL	82	138
MUTHU	60	M	140	100	24	NORMAL	NIL	0.5	NORMAL	230	148
MUTHUSAMY	58	F	150	90	19	NORMAL	NIL	0.7	NORMAL	220	182
SHAHIRABANU	60	F	140	100	34	NORMAL	NIL	1.4	LVH	84	186
MAHAMADHU	55	F	130	90	24	NORMAL	NIL	0.8	NORMAL	92	193
DHANALAKSHMI	47	F	180	120	35	NORMAL	NIL	0.9	LVH	98	195
LAKSHMI	61	F	130	90	21	NORMAL	NIL	1	NORMAL	90	180
JOTHIMANI	54	F	160	100	20	GRADE 1 HR	NIL	1	LVH	94	185
PANJAVARNAM	45	F	150	100	21	NORMAL	NIL	0.7	NORMAL	160	228
VIJAYA	50	F	130	90	27	NORMAL	NIL	0.8	NORMAL	140	160
IYYAMMAL	45	F	130	90	24	NORMAL	NIL	1	NORMAL	160	145
SELVARAJ	46	M	140	90	20	NORMAL	NIL	1	NORMAL	100	242
VASUDEVAN	64	M	150	90	24	NORMAL	1+	1	NORMAL	340	174
SUBRAMANIAN	65	M	140	90	22	NORMAL	NIL	1	NORMAL	86	170
NATRAJ	83	M	150	90	19	NORMAL	NIL	1	NORMAL	95	151
SUSEELA	57	F	210	120	19	GRADE 2 HR	2+	3.6	LVH	100	183
MOHAN	62	M	200	120	26	GRADE 1 HR	NIL	0.9	LVH	87	223
KANNAN	53	M	140	100	37	NORMAL	NIL	0.9	NORMAL	90	168
SEMBAI	50	F	140	90	20	NORMAL	NIL	0.8	NORMAL	110	221
LEELA	39	F	150	100	34	NORMAL	NIL	0.9	LVH	130	216
NASEERA	36	F	140	90	37	NORMAL	NIL	1.8	NORMAL	90	195
RUKKIYA	43	F	150	90	26	NORMAL	NIL	1.2	NORMAL	92	164
PADMA	57	F	140	100	21	NORMAL	NIL	1.2	LVH	140	236
KRISHNASAMY	57	M	170	100	22	GRADE 1 HR	NIL	1.2	LVH	130	194
VINCENT	57	M	140	100	19	NORMAL	NIL	1.1	NORMAL	86	248
MANJULA	50	F	170	100	25	NORMAL	1+	1	LVH	70	141
NAGAPPAN	54	M	160	100	19	GRADE 2 HR	NIL	1.2	LVH	80	165
SAGAYARAJ	58	M	160	110	27	GRADE 1HR	1+	1.2	LVH	200	150
ELANSELVI	33	F	140	100	26	NORMAL	NIL	0.8	NORMAL	69	113

KEY TO MASTER CHART:

SBP- systolic blood pressure	U/A- urine albumin
DBP- diastolic blood pressure	S.Cr- serum creatinine
BMI- body mass index	ECG- electrocardiogram
FBS- fasting blood sugar	S.TC- serum total cholesterol

CONSENT FORM

I have come to know that Dr. M.MANGAI SUSEELA, Postgraduate in the Department of General Medicine is conducting a study on the topic, "**A STUDY ON END ORGAN DAMAGE IN NEWLY DETECTED HYPERTENSIVE PATIENTS**". I understand that I will not have to suffer anyharmful consequences as a result of the study nor will I have any financial constraints. It is understood that blood will be collected from me for the purpose of conducting this study. I also understand that I can withdraw myself from this study at any point of time and by doing so it will not affect my treatment in any manner. Understanding all these, I wholeheartedly agree to take part in this study.

Signature Signature

Name of the patient: Name of the doctor:

Place :

Date :

ஒப்புதல்படிவம்

பெயர் :

வயது:

பாலினம்:

முகவரி:

கோவை அரசு மருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர் –தலைமையில்

நடைபெறும்இந்தஆய்வில்முழுசம்மதத்துடன் கலந்துகொள்ளசம்மதிக்கிறேன். இந்தஆய்வில்என்னைபற்றி விவரங்களைபாதுகாப்புடன்இந்தஆய்வில்வெளியிட ஆட்சேபணைஇல்லைஎன்றுதெரிவித்துக்கொள்கிறேன். எந்தநேரத்திலும்ஆய்வில்இருந்துவிலக்கிக்கொள்ளும் உரிமைஉண்டுஎன்று அறிவேன்.

இடம்:

தேதி:

கையொப்பம் / ரேகை